Diseases of Swine

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DISEASES OF SWINE

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PREFACE

FORTY-EIGHT AUTHORS have devoted themselves to a common cause in this volume: the publication of a systematic arrangement of complete, accurate, and most up-to-date information on Diseases of Swine. Every effort has been made to present the facts and theory in a logical manner for direct application in veterinary practice, veterinary schools, and in research. Emphasis has been placed on accuracy and thoroughness.

Both the practitioner and the student should find the cross-referencing and arrangement of chapters easy to use. Uniformity of chapter arrangement has been stressed within the limits of comparative subject matter, while at the same time preserving authors' individual styling. A definite attempt has been made to keep duplication at a minimum without disrupting subject continuity and clarity within a given chapter.

Detailed information has been included for those who wish to explore more deeply into the etiology, pathogenesis, diagnosis, treatment, and prevention of diseases of swine. The extensive reference lists offer the interested readers a key to still further study.

A broad, objective approach to the many diseases discussed has been achieved through the willing cooperation of eight prominent scientists and educators serving as an Advisory Board. These men agreed to draw upon their wide experience and knowledge to review specific areas in which they were particularly well informed. This had the effect of arbitrating controversial issues and insuring balance for the information presented.

It is believed that all who are interested in swine diseases will find this text to be a most complete and authoritative reference on the diseases of importance to the swine raisers of North America.

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Anatomy and Physiology

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CHAPTER 1

Anatomy

The pig, *Sus scrofa,* belongs to the super order UNGULATA with the other hoofed mammals The four digits place it in the order of even toed hoofed animals ARTIODACTYLA

SKELETON

Teeth

The pig possesses the standard number of teeth In the permanent dentition there are 3 incisors, 1 canine, 4 premolars, and 3 molars on each side of the jaw above and below The total is 44 In the temporary dentition there are 3 incisors, 1 canine, and 3 molars on each side above and below, making a total of 28 Each permanent incisor and canine tooth replaces the corresponding deciduous tooth The deciduous molars are replaced by the posterior 3 premolars No teeth precede the permanent molars

The upper and lower incisor areas are shaped so that the medial teeth lie in a plane decidedly anterior to the lateral teeth The upper central incisor is oval in cross section and angles sharply downward and medially The intermediate incisor bends medially and lies slightly posterior to the central incisor A space separates the intermediate incisor from the small corner incisor

The lower incisors are close together (especially 1 and 2) They are elongate and project anteriorly The intermediate

tooth is slightly larger than the central and much larger than the corner incisor

There is an interval between the canine tooth and the corner incisor especially in the upper jaw The canine tooth (tusk) is large, especially in the boar, and projects outside the mouth The upper canine is posterior to the lower They wear against each other, maintaining a sharp edge The upper tooth is oval in cross section, the lower is triangular The pulp cavity remains throughout the life of the tooth allowing it to continue to lengthen

The cheek teeth increase in size from front to back They are bunodont in type since their multiple cusps are moundlike The crowns are short, forming a neck near the roots. The table surfaces of the molars consist of complex crushing mounds while those of the premolars are simple cutting areas The first premolar in each jaw is small and simple The one in the mandible lies just caudal to the canine tooth, whereas the upper one is separated from the canine tooth by a space This space in the lower jaw is between the first and second premolars In the upper jaw the first and second premolars possess 2 roots, the third 3, and the fourth 5 The molars have 6 roots In the lower jaw the first premolar has 1 root, the second and third 2, and the fourth 3 Four roots are possessed by the first and second molars and 5 by the third

The upper deciduous molars possess 2, 3, and 4 roots, respectively The lower deciduous molars have 2 roots, except the last one which has 5 The deciduous teeth tend to resemble the permanent teeth that replace them The last lower one, however, is different in that it possesses 8 pairs of cusp units

The lateral incisors and the canines are present at birth The deciduous molars and central incisors erupt during the first month The intermediate deciduous in
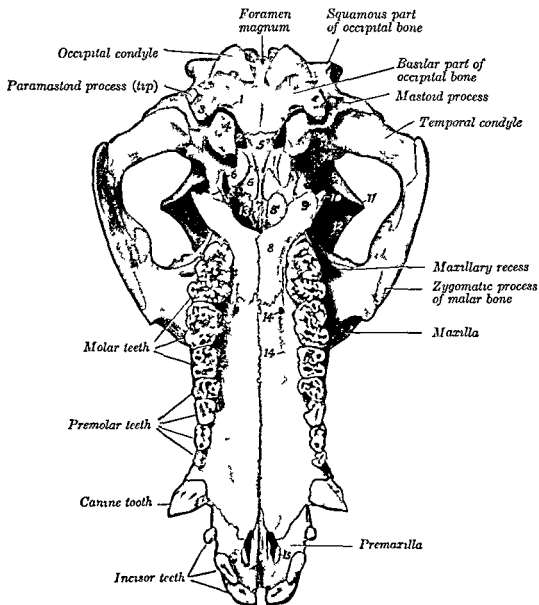
FIG 1 1—Skull of pig, ventral view, without mandible and hyoid (From Sisson and Grossman, 1953   Courtesy W B Saunders Co)

1 Hypoglossal foramen	8 Perpendicular part of palatine bone
2 Foramen lacerum anterius	9 Pterygoid process of palatine bone
3 Foramen lacerum posterius	10 Pterygoid process of sphenoid bone
4 Bulla tympanica	11 Supraorbital process
5 Body of sphenoid	12 Orbital opening of supraorbital canal
6 Pterygoid bone	13 Choanae or posterior nares
6' Hamulus of pterygoid bone	14 14' Anterior palatine foramen and groove
7 Vomer	15 Palatine fissure
8 Horizontal part of palatine bone	

cisors appear after 2 months The first pre molars and first permanent molars appear at 5 months The permanent corner incisors and the canines erupt at about 9 months The permanent central incisors and second molars erupt at about 12 months By 15 months the last 3 premolars have appeared The last molars have erupted by 18 months

The placement of enamel, dentine, and cementum is like that of an ordinary simple tooth, except on the permanent canine where enamel is on the convex surface and cementum is on the concave surface

The occlusal surfaces of the cheek teeth form a straight line when viewed from the side The upper premolars are slightly lateral to the lower ones in position The distance between the cheek teeth of the right and left sides is less posteriorly than anteriorly The upper and lower corner incisors usually do not contact each other

Axial Bones

The vertebral formula is C 7, T 14–15, L 6–7, S 4, Cy 20–23 The cervical region is short The dorsal spines of the cervical vertebrae are tall, as are those of the thoracic area The arch in the cervical and thoracic regions is perforated by a foramen which is in addition to the invertebral notches The lumbar transverse processes do not articulate with each other or with the sacrum The vertebrae composing the sacrum do not fuse to the extent that their identity is lost Their dorsal spines are almost absent The lumbosacral space is medium in size The first coccygeal vertebra often fuses with the sacrum

The ribs are strongly curved, making a long barrel shaped thorax Seven are sternal and 7 or 8 asternal The fifteenth rib, when present, is often floating in type

The sternum is flat, especially posteriorly, and consists of 6 sternebrae The first segment projects forward and is flattened laterally

Skull

The skull is massive The long and narrow nasal and frontal areas which are straight in young animals become dished later This is especially true in the more brachycephalic breeds The nuchal crest is very prominent and the temporal fossa is entirely lateral The external acoustic process is dorsal in position in respect to the posterolateral areas and projects dorso laterally The supraorbital process does not contact the heavy zygomatic arch The round and orbital foramina are combined as the foramen orbitorotundum The maxillary foramen is large There is usually a prominence over the lateral side of the alveolus of the upper canine tooth A short three sided prism, the os rostri lies in the nasal septum between the anterior portions of the nasal and premaxillary bones The paramastoid processes are extremely long and the bulla tympanica is prominent The jugular and oval foramina are in the form of a long slit, the foramen lacerum, medial to the bulla The elongate but small posterior nares are divided vertically by the vomer Each palatine bone forms a tuberosity which projects ventrally posterior to the last molar The palate is long and narrow even in the shorter skulls It is widest in the area between the canine teeth There is a distinct fossa posterior to the central incisors, associated with the incisive foramina The cranial cavity is relatively small and separated from the frontal surface by a spacious frontal sinus The pituitary fossa is deep The dorsal turbinate is long, narrow, and unscrolled and projects downward from the nasal bone to lie slightly medial to the large double scrolled ventral turbinate The frontal sinus increases in size as the animal matures It extends from slightly behind the level of the infraorbital foramina to the posterior limit of the skull The nuchal area is usually solid but may be undermined next to the cranial cavity by the sinus The right and left sinuses are separated, each being further divided by numerous incomplete septa Anteriorly there is communication with the ethmoidal meatuses The small maxillary sinus occupies the area medial to the anterior attachment of the zygomatic arch, above the

infraorbital canal The roots of the molars do not project into it It communicates with the middle meatus of the nasal cavity The body and wings of the sphenoid bone are excavated to form the relatively large sphenoidal sinus It communicates anteriorly with the ventral ethmoidal meatus

The right and left sinuses tend to be separated in the midline The perpendicular part of the palatine bone may also form a part of the sinus

The mandible is strong and massive The body is pointed anteriorly, concave dorsally, and convex ventrally The right
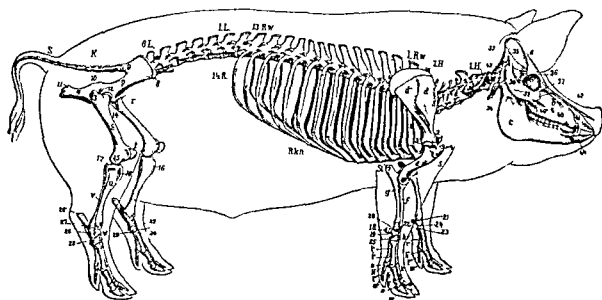
FIG 1 2—Skeleton of pig lateral view (From Ellenberger, in Leisering s Atlas)

a *Cranium*
b *Maxilla*
c *Mandible*
1H 7H *Cervical vertebrae*
1R w *First thoracic vertebra*
13R w *Thirteenth thoracic vertebra (next to last)*
3L *First lumbar vertebra*
6L *Sixth lumbar vertebra (next to last usually)*
K *Sacrum*
S *Coccygeal vertebrae*
1R *First rib*
14R *Last rib*
R kn *Costal cartilages*
St *Sternum*
d *Supraspinous fossa*
d *Infraspinous fossa*
1 *Spine of scapula*
2 *Neck of scapula*
e *Humerus*
3 *Head of humerus*
4 *Tuberosities of humerus*
5 *Deltoid tuberosity*
6 *Lateral epicondyle of humerus*
f *Radius*
g *Ulna*
7 *Olecranon*
h *Carpus*

18 25 *Carpal bones*
i 1″″ *Metacarpus*
k k ″ *Proximal phalanges*
11″″ *Middle phalanges*
m m″″″ *Distal phalanges*
n o *Sesamoids*
p *Ilium*
8 *Tuber coxae*
9 *Tuber sacrale*
10 *Superior ischiatic spine*
q *Ischium*
11 *Tuber ischii*
r *Pubis*
12 *Acetabulum*
s *Femur*
13 *Trochanter major*
14 *Trochanter minor*
15 *Lateral epicondyle*
t *Patella*
u *Tibia*
16 *Crest of tibia*
17 *Lateral condyle of tibia*
v *Fibula*
w *Tarsus*
26 31 *Tarsal bones*
26 *Tuber calcis*
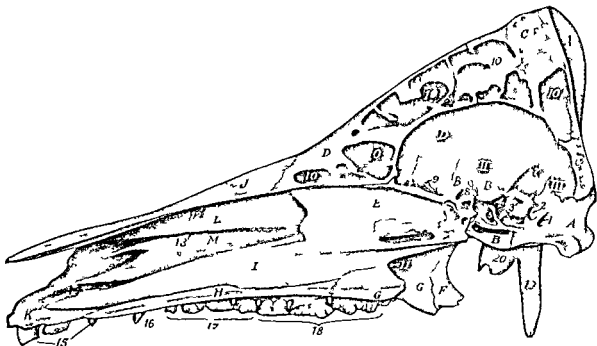
FIG 1 3—Sagittal section of skull of p g without mand ble (From S sson and Grossman 1953   Courtesy W B Saunders Co)

A A   Bas lar and squamous parts of occip tal bone
B   Body of spheno d bone
B   Temporal wing of sphenoid bone
B   Orb tal wing of spheno d bone
C   Parietal bone
D D   Internal and external plates of frontal bone
E E   Cr briform and perpend cular plates of eth mo d bone
F   Pterygoid bone
G G   Perpend cular and hor zontal parts of pala t ne bone
H   Palat ne process of maxilla
I   Vomer
J   Nasal bone
K   Body of premax lla
L   Dorsal turb nate bone
M   Ventral tu b nate bone
I II III   Fossae cran i
1   Hypoglossal foramen

2   Foramen lacerum poster us
3   Meatus acust cus nternus
4   Foramen lacerum anter us
5   Hypophyseal or p tu tary fossa
6   Foramen orbito rotundum
7   Later al crest between cerebral and cerebellar parts of cran al cav ty
8   Opt c foramen
9   Ethmo dal foramen
10   Frontal s nus
11   Meatus nasopharyngeus
12 13 14   Dorsal m ddle and ventral nasal meatuses
15   Inc sor teeth
16   Can ne tooth
17   Premolar teeth
18   Molar teeth
19   Paramasto d process
20   Bulla tympan ca

and left portions are fused The horizontal rami are thick and contain several mental foramina The mandibular canal is large The condyle is convex in all directions and is situated posterior to a short coronoid process

The hyoid bone consists of a flat body basihyoid which continues directly back ward as the wide curved thyrohyoids The epihyoids and stylohyoids are slender The tympanohyoids are thin and cartilaginous The keratohyoids are very short

Limbs

The bones of the limbs are relatively massive The scapula is very wide at its vertebral border Its prominent spine pos sesses a large tuberosity but only a rudi mentary acromion The lateral tuberosity of the humerus is very large and projects anterior to the single bicipital groove The large ulna is not fused with the radius and continues to the carpus There are 8 carpal bones 4 in each row Four metacarpal

bones are present Each of the four digits consists of 3 phalanges The abaxial digits are shorter and smaller than the axial ones A pair of proximal sesamoid bones rests on the volar surface of the distal portion of each metacarpal bone A distal sesamoid bone is present in each axial digit

The ilia are parallel to each other and tip forward, producing a very sloping pelvic inlet The superior ischiatic spines

FIG 1 4—Mandible of pig dorsal view (From Sisson and Grossman, 1953 Courtesy W B Saunders Co)

A Body
B B' Horizontal and vert cal parts of ramus
C Condyle
D Coronoid process
1,2,3 Incisor teeth
4 Can ne tooth
5 6,7 Premolar teeth (first absent)
8 9 10 Molar teeth

are prominent and increase the concavity of the pelvic floor The symphysis is rather thick and not firmly fused The floor of the pelvis slopes more posteriorly, the symphysis is thinner, and the ischial tubera are more everted in the female The rim of the acetabulum is thick and notched posteriorly The trochanter major of the femur is single The supracondyloid fossa and the third trochanter are absent The patella is thick anteroposteriorly The tibia is typical The fibula is large and extends to the tarsus, which consists of 7 bones The articular surfaces are placed so that movement occurs not only between the tibial tarsal bone and the tibia but also between the tibial tarsal bone and those adjacent to it The metatarsals and phalanges are like those of the forelimb except that they tend to be slightly longer There is an extra sesamoid bone behind the proximal portion of the medial axial metatarsal bone

The epiphyseal lines do not completely disappear from the vertebral bodies and the long bones for several years

RESPIRATORY SYSTEM

Nasal Cavity

The snout, or rostrum is a cylindrical projection with a prominent margin It is practically hairless and is smooth and fuses with the upper lip The nostrils are small The anterior extremity of the nasal septum is ossified as the os rostri Cartilages tend to form the framework of the nostrils and to fill in the nasomaxillary notch

The nasal cavity is long and narrow except in short nosed breeds The long round posterior nares are separated from the upper posterior part of the cavity by a transverse lamina and from each other by the vomer The dorsal turbinate is thin anteriorly but gradually increases in diameter posteriorly It projects ventrally and medially from the dorsolateral wall of the nasal cavity so that its ventral edge lies medial to the dorsal part of the ventral turbinate The ventral turbinate is much larger than the dorsal turbinate and begins
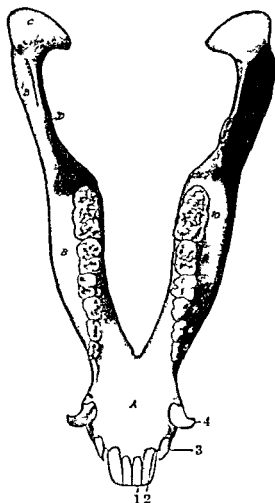
anteriorly from a fold which projects from the lateral wall of the cavity just behind the nostril The passageway from the nostril is thus somewhat obstructed except dorsally The scrolls of the ethmoid area do not project forward as a middle turbinate The dorsal and middle meatuses are very narrow The ventral meatus is somewhat larger especially posteriorly where the ventral turbinate becomes wrinkled longitudinally A small opening in the posterolateral part of the middle meatus communicates with the maxillary sinus dorsal to which are several small openings to the frontal sinus, via the ethmoidal meatuses In the posterolateral part of the ventral meatus is the opening of the nasolacrimal duct

The anterior or vestibular region is lined with a stratified squamous epithelium This changes gradually into a stratified columnar and then a ciliated pseudostratified columnar epithelium with goblet cells in the main or respiratory area The olfactory mucosa is brown and thick and contains special cells

Larynx

The larynx is relatively large and does not articulate with the hyoid bone The epiglottis is very large, broad anteriorly and loosely attached to the rest of the larynx The arytenoid cartilages are extensive dorsoventrally The rima glottidis is narrow A long vertical slit associated with the vocal fold opens into the large saccule

Pleura

The long rounded thorax is lined by pleura of medium thickness The pleural sacs do not project forward beyond the first rib on the right and the first intercostal space on the left The diaphragmatic reflection follows the costal attachment of the diaphragm Since the mediastinal pleura and the caval fold are not perforated the two pleural cavities are entirely separate

Lungs

The lungs are divided into lobes and lobules The latter however are not as distinct as those of the ox The apical lobe of the right lung is often double and is provided with a special divided bronchus The right lung also possesses an intermediate lobe The left lung has the standard apical cardiac and diaphragmatic lobes The cardiac notch which is in the shape of an inverted V, is between the cardiac and apical lobes The left notch extends further dorsally and is slightly longer The right one extends from the second intercostal space to the fifth rib and the left one from the second rib to the fifth rib or intercostal space

Diaphragm

lapping The larger bronchi have irregular plates of cartilage in their walls the smaller ones have none

DIGESTIVE SYSTEM

Oral Cavity

The hard palate is long and narrow especially in long nosed breeds It is slightly narrower posteriorly than anteriorly The many transverse ridges are not continuous in the midline and may alternate in position anteriorly The incisive papilla with the two incisive openings is located in the median plane Lymphoid or tonsillar tissue is found on the soft palate the lateral walls of the isthmus faucium and the root of the tongue
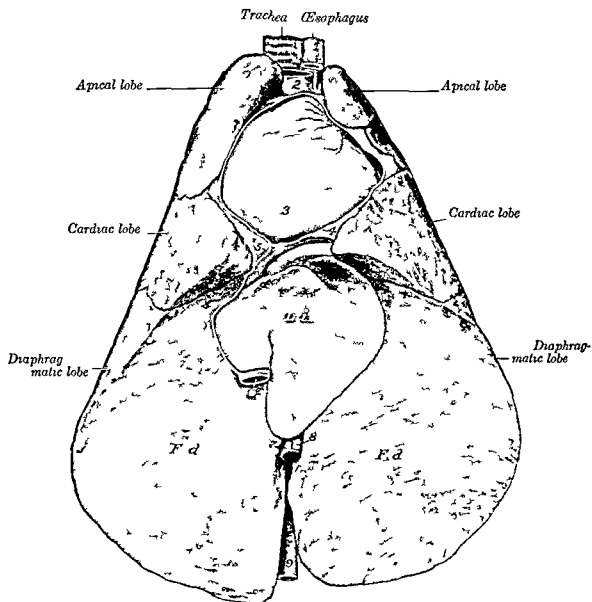
FIG 1 5—Lungs and heart of pig ventral view (From Sisson and Grossman, 1953 Courtesy W B Saunders Co)

L I Intermediate lobe of right lung
F d Diaphragmatic surface of lungs
1 Left brachial artery (subclavian)
2 Brachiocephalic artery
3 Apex of heart
4 Pericardium (cut edge)
5 Plica venae cavae
6 Posterior vena cava
7 Esophagus
8 Ventral esophageal nerve trunk
9 Aorta
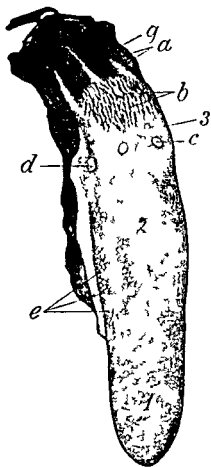
FIG 1 6—Tongue of pig (From Ellenberger and Baum 1943)

1 Apex
2 Dorsum
3 Root
a O fces of ducts of l ngual glands
b Pap llae of root
c Vallate pap lla (not really so d st nct as n f gure)
d Fol ate pap lla
e Fung fo m pap llae
f Ep glott s (pulled back)
g Med an glossoep glott c fold

The long narrow tongue possesses m ny long pointed papillae on its dorsal surface at the root Fungiform papillae are nu merous over the entire surface A small group of foliate papillae is found postero laterally Two or three vallate papillae are located on the posterior part of the dorsal surface The frenum linguae is double The median septum contains a cord of fat
The teeth are discussed with the skull

Salivary Glands

The large salivary glands are serous mucous or mixed in type and have a com pound tubuloalveolar organization The small branched tubuloalveolar glands of the oral submucosa occur singly or in dense groups The buccal group is composed of mucous and serous units Serous glands are found beneath the smooth surface of the upper lip below the nostrils

The parotid gland is very extensive forming a triangle with its base downward ventral to the ear posterior to the masseter muscle The duct courses ventral to the masseter and empties into the oral cavity lateral to the first upper molar The gland is serous in type

The mandibular (submaxillary) gland is smaller and darker than the parotid gland and round in outline its thickness being almost as great as its diameter It lies under cover of the lower part of the parotid gland A portion extends forward for a short distance along the duct which in turn courses along the medial surface of the mandible deep to the mylohyoideus muscle to empty into the oral cavity near the frenum linguae The gland is mixed in type

The sublingual gland consists of two parts which are distributed along the mandibular duct The posterior part is long (5 cm) and flat and lies just anterior to the mandibular gland Its duct or ducts accompany and open with the mandibular duct The anterior portion is larger hav ing 8 to 10 ducts which open directly into the oral cavity It is mixed in type

Pharynx

Dorsal to the beginning of the esophagus is a diverticulum which extends posteriorly for about 3 cm The nasopharynx which is above the soft palate bears a ciliated pseudostratified epithelium with goblet cells The oropharynx or isthmus faucium which is ventral to the soft palate is lined with a stratified squamous epithelium The mucosal glands are mucous in type in the oropharynx and mixed in the nasopharynx

Esophagus

The esophagus is especially dilatable at its ends It tends to lie on the left of the trachea at the thoracic inlet The muscle
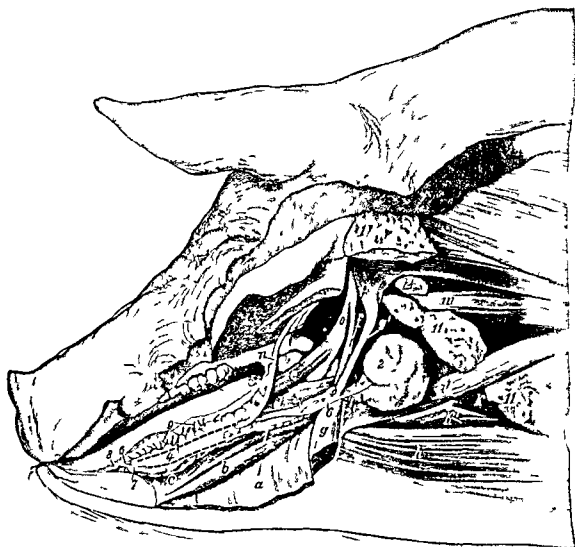
FIG 1 7—Dissection of mouth and pharyngeal reg on of pig (From Ellenberger and Baum, 1943)

1 Dorsal end of parot d gland
2,2 Mand bular gland
3 4 Posterior and anter or parts of subl ngual gland
5 Palat ne glands
6,6 Mand bular duct (dotted part concealed)
7,7 Ductus subl ngual s major (dotted part con cealed)
8 Opening of 6 and 7
9 Ductus subl nguales minores
10 Tons l
11 Thymus
12 Pharyngeal lymph gland
a. M. mylohyo deus (reflected)

b M gen ohyo deus
c M. genioglossus
d M hyoglossus
e M. styloglossus
f M stylohyoideus
g M d gastricus (cut)
g Tendon of origin of d gastricus
h M. sternohyoideus
I M. omohyo deus
k k M sternothyro deus
m M. rectus capitis ventralis major
n Lingual nerve
o Great cornu of hyo d bone
p Paramastoid process

layers are striated except near the cardia
Mucous glands are present in the sub
mucosa, especially in the cervical portion
Lymph nodules are also numerous The
epithelium is stratified squamous

Stomach

The stomach lies transversely with the
greater curvature directed ventrally The
stomach of a large animal may hold as
much as 8 liters The left portion beyond
the cardia, possesses a posteriorly pointing
diverticulum (diverticulum ventriculi) A
constriction, which is especially evident in
ternally, separates it from the main portion
of the stomach The esophagus joins the
stomach obliquely at the left end of the
lesser curvature The mucosa of the
stomach near the cardia is of the esoph
ageal type The whole left area and the
diverticulum are pale gray and constitute

the cardiac gland region The mucosa of
the fundic gland area which is thick and
reddish brown in appearance, does not
quite reach the lesser curvature The py
loric extremity contains the pyloric glands
It is pale and interrupted by low folds
The gland types blend with each other As
the pylorus constricts to become contin
uous with the duodenum, it possesses on
its lesser curvature a fatty, fibrous, knob
like prominence (torus pyloricus), which
protrudes into the lumen and diminishes
the size of the orifice

Intestinal Tract

The small intestine is about 18 M in
length The duodenum passes posteriorly
on the right side, swinging medially and
dorsally to pass posterior to the mesenteric
vessels Here it is close to the dorsum As
it turns forward to become the jejunum it
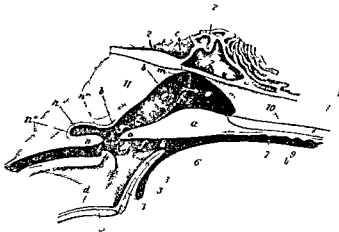
FIG 1 8—Sagittal section of pharyngeal region of pig partly schematic (From Ellen
berger in Leisering s Atlas)

1 Palatine bone
2 Sphenoid bone
2 Sphenoidal sinus
2 Occipital bone
3 Epiglottis
4 Arytenoid cartilage
5 Thyroid cartilage
6 Root of tongue
7 Mouth cavity
8 Isthmus faucium
9 Hard palate
10 Septum nasi
11 Ventral muscles of head
a Soft palate

a Free edge of soft palate
b Dorsal wall of pharynx
c Fornix of pharynx
d Cavity of larynx
e g Nasopharynx
f Oropharynx
h Posterior pillar of soft palate
Dotted line indicating lateral boundary between
nasal cavity and pharynx
k Aditus laryngis
l Aditus oesophagi
m Eustachian orifice
n Pharyngeal diverticulum
o Posterior naris

is in contact with the ventromedial surface of the terminal colon. The mesentery of the duodenum is short. The duodenum is represented by approximately the first 60 cm of the small intestine. The submucosal glands extend along the first part of the small intestine for 3–4 M. The bile duct opens into the duodenum 3–5 cm. from the pylorus The orifice of the pancreatic duct is 10 cm. beyond that of the bile duct. The jejunum and ileum are suspended from the sublumbar area by a short (20 cm.) mesentery. They lie against the right posterior abdominal wall and above the coils of the large intestine. The ileum follows the dorsal surface of the cecum to which it is connected by the ileocecal fold of peritoneum. It opens into the cecum acutely, forming a distinct valve where the cecum is directly continuous with the colon. This occurs behind and to the left of the root of the mesentery Aggregated and solitary lymph nodules are numerous in the mucosa and submucosa, except in the anterior part of the duodenum.

The large intestine is 4–5 M. in length. The cecum is 20–30 cm. long and 7–10 cm. in diameter. Its blunt apex lies in the mid-line and points toward the right. The apex is usually the most posterior part of the mass of large intestines. It courses forward and upward on the left, where it continues directly as colon. The colon forms coils about the colic branches of the mesenteric vessels. These coils occupy the left and anterior areas of the abdominal cavity, extending as far forward as the stomach. Beginning at the cecocolic junction, which is on the left, the colon continues forward and then to the right, making about three clockwise turns, each being ventral to the one which precedes it. When the floor of the cavity is reached, the direction is reversed and the colon ascends in a counter clockwise direction, alternating with the previous turns, passing them on their concave surfaces. The last and most dorsal turn passes from right to left anterior to the anterior mesenteric artery as the transverse colon, continuing on the left dorsal wall as the terminal colon and terminating as the rectum The peritoneum is absent between the adjacent coils of the colon. The diameter of the gut is less in the returning portions of the coils. There are two longitudinal bands of muscle with intervening sacculations in the first parts of the colon. The cecum has three bands and three series of sacculations. Solitary lymph nodules are numerous throughout the large intestine. Embedded in the lymphoid tissue in the submucosa of the colon are numerous branched, tubular mucous glands.

Liver

The liver is relatively large (1.5–2 kg.). It is convex anteriorly and concave posteriorly, having thin edges but a thick central portion. It lies between the diaphragm and the stomach. The incisures between the lobes are not deep The lobes are designated as caudate, right lateral, right central, left central, and left lateral. The left lateral lobe is usually the largest. The right lateral and caudate lobes extend farthest posteriorly. They are not indented for the right kidney, however, except in the very young animal. The posterior vena
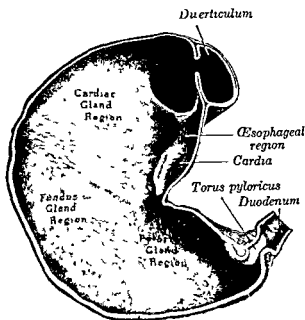
FIG. 1 9—Frontal section of stomach of pig. (From Sisson and Grossman, 1953. Courtesy W. B Saunders Co)

cava runs within the left edge of the right lobes. To the left of this is an esophageal notch. The gall bladder is somewhat imbedded in the visceral portion of the right central lobe. The cystic duct joins the hepatic duct at an acute angle at the portal fissure. The bile duct enters the duodenum 3–5 cm. from the pylorus. The portion of that lobe which is medial to the gall bladder is sometimes named the quadrate lobe. The papillary process is not prominent. The peritoneal ligaments are represented only by a coronary and a small falciform ligament. The round ligament thus travels independently to the umbilical area. The lobules are distinct.

Pancreas

The pancreas lies in the mesoduodenum and the greater omentum, and is thus situated transversely across the dorsal wall of the abdominal cavity behind the stomach and in front of the root of the mesentery. The right portion, which lies next to the duodenum, is larger than the left portion, which is related to the spleen, gastric di-
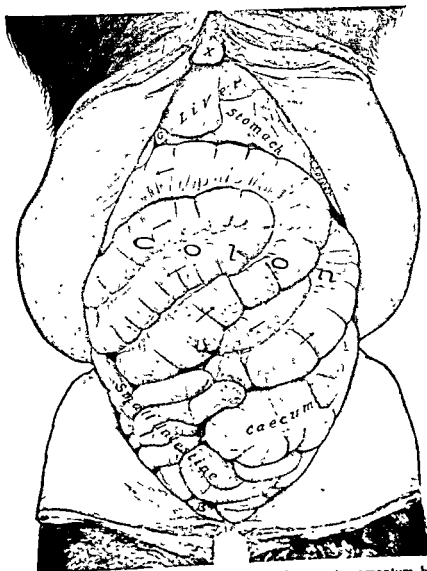
FIG. 1.10—Abdominal viscera of pig; ventral view. The greater omentum has been removed. Arrows indicate course of coils of colon. The spleen was contracted. (From (Sisson and Grossman, 1953. Courtesy W. B. Saunders Co.)

B. Urinary bladder   X. Xiphoid cartilage
G. Gall bladder
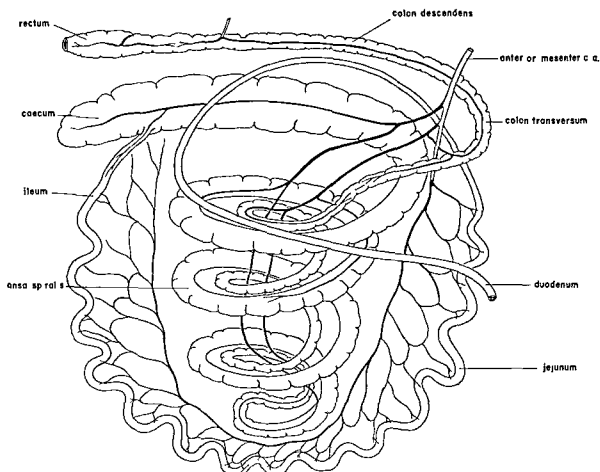
FIG 1 11—Schema of intestinal tract of pig (From Getty, 1955 Courtesy Burgess Publishing Co and R Getty)

verticulum and left kidney The lobules are distinct The duct leaves the right portion to enter the duodenum about 15 cm from the pylorus The islets are not easily discernible microscopically

Spleen

The spleen is long (50 cm), narrow, and flat with tapered ends The size varies greatly, however It lies vertically in the greater omentum on the left part of the greater curvature of the stomach The splenic vessels course down its medial surface

Peritoneum

The peritoneum is medium in thickness as compared with that of other animals It extends into the pelvic cavity in the form of pouches which end before they reach the posterior wall The rectogenital pouch is the largest The vesicogenital and vesicopubic pouches do not extend as far poste

riorly The lateral and ventral ligaments of the urinary bladder extend to the umbilicus in the young pig, but their anterior portions disappear in the adult The mesentery for the coils of the colon and for the jejunum and ileum arise in the lumbar area, enclosing the origin of the anterior mesenteric artery The lesser omentum is short, but the greater omentum is extensive and lacelike It may lie on the abdominal floor below the more anterior portions of the intestinal coils It attaches to the dorsum at the area occupied by the transverse colon The epiploic foramen is in the characteristic place between the posterior vena cava and the portal vein

URINARY SYSTEM

The kidneys are not lobate externally They are bean shaped, flattened dorsoventrally, and somewhat pointed at the anterior and posterior poles They lie

beneath the first four lumbar vertebrae but the left kidney is usually slightly more anterior than the right one which thus does not contact the liver in the adult Each kidney has a fibrous capsule which is covered by a large deposit of fat that extends into the renal sinus between the calyces and large vessels The hilus is represented by an indentation on the medial surface, which leads to the renal sinus The latter contains the enlarged origin of the ureter the pelvis The medulla consists of about 20 pyramids with a minor calyx fitted around the apical half of each The pelvis receives two major calyces each of which is formed by the confluence of minor calyces Several papillary ducts open on the papilla of each pyramid Renal columns are present between the pyramids The loops of Henle are very long The kidney in the adult measures about 6 by 13 cm and weighs

200 250 gm Their combined weight constitutes about 1/200 to 1/150 of the body weight

The renal artery arises from the aorta and passes through the ventral part of the hilus dividing into interlobar branches between the pyramids At the cortico medullary junction they bend at nearly right angles to become the subcortical (arcuate) arteries From these interlobular arteries course between the medullary rays to supply the capsule and the afferent arterioles of the glomeruli The efferent arterioles leave the glomeruli close to the afferent ones and form capillary beds about the uriniferous tubules The efferent arterioles also give branches arteriolae rectae which enter the medulla to supply the pyramids The capillary beds of the cortex and medulla empty into the interlobular veins From this point the veins closely accompany the arterial tree
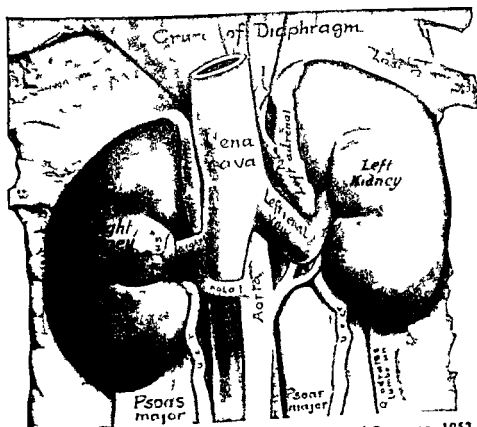
FIG 1 12—Kidneys of pig in situ ventral view (From Sisson and Grossman 1953 Courtesy W B Saunders Co)

1 Hepatic artery
2 Gastrosplenic artery
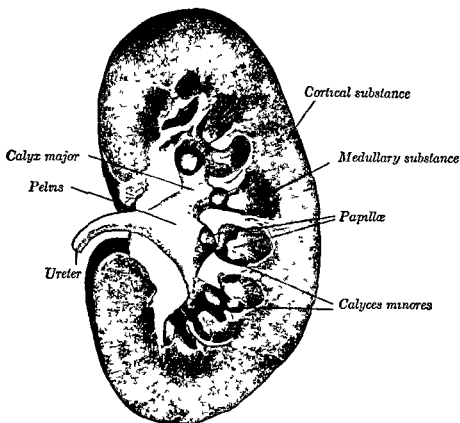
FIG. 1.13—Frontal section of kidney of pig. (From Sisson and Grossman, 1953. Courtesy W. B. Saunders Co.)

The ureter, large at first, begins at the pelvis of the kidney leaving the dorsal part of the hilus to course in a slightly flexuous manner through the sublumbar fat to the urinary bladder near its neck. The bladder is relatively large and projects decidedly into the abdominal cavity. The peritoneum covers it dorsally as far back as the openings of the ureters. The urethra is discussed with the genital system. The urinary epithelium is transitional in type.

MALE REPRODUCTIVE ORGANS

Testes

The large testicles are contained in a non-pendulous scrotum located in the posterior area below the anus. The tail of the epididymis is dorsal and the head ventral. The mesorchium is next to the animal's body throughout its extent. The body of the epididymis is contained in a fold from the lateral portion of the mesorchium in contact with the testicle. The ductus deferens is in a fold of mesorchium extending medially. The parietal layer of tunica vaginalis (tunica vaginalis communis) forms the lining of the scrotum and is continuous with the peritoneum at the internal inguinal ring, where it forms the vaginal ring. The visceral layer of tunica vaginalis (tunica vaginalis propria) is the outer layer of the cord and testicle. Between the two layers is the cavity of the tunica vaginalis, which is continuous at the vaginal ring with the peritoneal cavity. Directly beneath the tunica vaginalis propria is the tunica albuginea, a heavy, dense connective tissue from which trabeculae extend into the glandular areas of the testicle to form its framework. The seminiferous tubules are disposed as lobules. Straight tubules from these converge at the mediastinum testis to form a network, the rete testis, from which several efferent ductules proceed to the head of the epididymis, where they independently join its duct. Interstitial cells are numerous. The testes are relatively large, having a combined weight in relation to body weight of 1:250 in the

adult. The epididymis if stretched out would measure more than 100 M. The testicles have descended by the time of birth.

Spermatic Cord

The external cremaster muscle, which lies just outside the tunica vaginalis communis, is large. The spermatic cord is long due to the posterior position of the testis It includes the spermatic artery, vein, nerves, lymphatics, and the associated ductus deferens covered by tunica vaginalis propria.

Penis

The penis measures, when extended, at least 50 cm. It is small in diameter (1–1 5 cm), however. The erectile tissue of the urethra does not expand to form a glans. The anterior extremity of the penis is pointed and spirally twisted, containing the slitlike opening of the urethra. The erectile tissue of the body of the penis is small in amount and the connective tissue abundant. There is a sigmoid flexure. The short, thick bulbocavernosus muscle is situ-
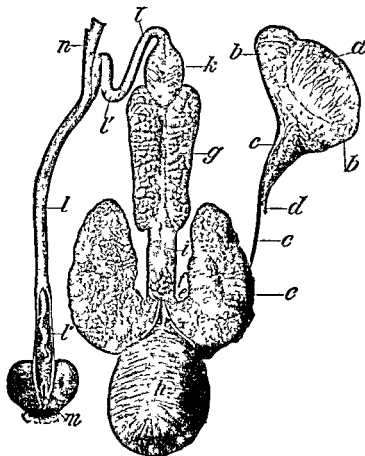
FIG. 1.14—Genital organs of boar The vesiculae seminales are drawn outward to show the structures which, in the natural position, are covered by them. (From Ellenberger and Baum, 1943.)

a Testicle
b Epididymis
c Ductus deferens
d Spermatic artery
e Vesicula seminalis
e′. Excretory ducts of vesiculae
f. Body of prostate
g Bulbo-urethral gland
h Urinary bladder
i Urethral muscle
k Bulbo-cavernosus muscle
l Penis
l′ Sigmoid flexure of penis
l″ Spiral anterior part of penis, exposed by slit-ting open prepuce
m. Orifice of preputial pouch
n Retractor penis muscle

ated near the ischial arch. The two retractor penis muscles join the ventral surface of the penis just anterior to the sigmoid flexure. The penis lies well within the prepuce in the quiescent state.

Prepuce

The prepuce has a long cavity and a narrow orifice. From the dorsal surface just posterior to the orifice is an opening to a large diverticulum, which is partially divided into two compartments by a median septum. It contains epithelial casts and urine. The preputial wall contains protractor muscles.

Urethra

The pelvic urethra is about 20 cm. long and is surrounded by a thick urethral muscle except dorsally, where it is fibrous. At the root of the penis is a distinct bulb. Projecting into the lumen from the roof of the urethra near the neck of the urinary bladder is the colliculus seminalis. Close to the midline on the latter are the ejaculatory orifices. Many glands are present throughout the submucosa.

Accessory Glands

Situated on the sides of the pelvic urethra toward the ischial arch are the long, cigar-shaped bulbourethral (Cowper's) glands. They are about 15 cm. in length and 3 cm. in diameter and contain much fibrous tissue and striated muscle in their walls. A large excretory duct leaves the medial surface of each gland posteriorly to open into the urethra close to the ischial arch. The gland is compound, consisting of mucous tubuloalveolar units. The secretion, however, is very thick, waxy, and tenacious.

The seminal vesicles are very large pyramidal masses (15 × 7 cm.) which lie dorsal to the neck of the urinary bladder. They tend to be blunt anteriorly and pointed posteriorly. They have lobate surfaces and a thin capsule. The medial surfaces are in apposition with each other. Several ducts unite to form a single large collecting sinus, which empties with or lateral to the ductus deferens at the ejaculatory orifice. The secretion is gray and watery.

The multilobar prostate gland consists of a small flattened body (3 × 4 × 1 cm.),

FIG. 1.15—Scrotum and testicle of boar. (From Getty, 1955. Courtesy Burgess Publishing Co. and R. Getty.)

1 Superficial inguinal lymph node
2 Penis
3 Testicle covered by tunica vaginalis
4 Spermatic cord
5 Cut edge of scrotum
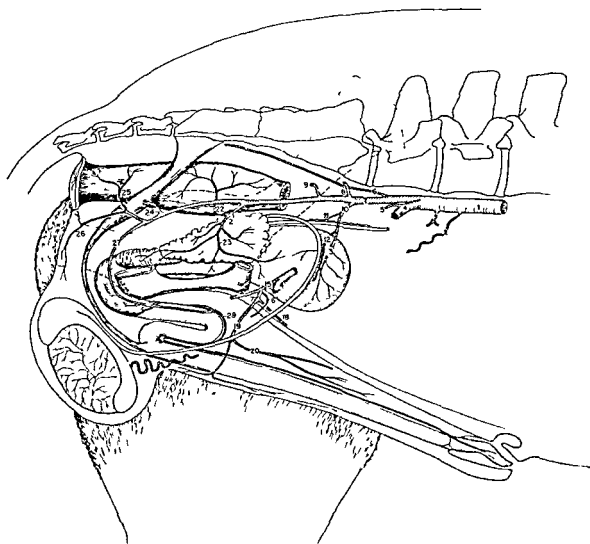
FIG 1 16—Blood supply to male genitalia of boar. (From Getty, 1955 Courtesy Burgess Publishing Co and R Getty)

1 Aorta
2 Internal spermatic artery
3 Posterior mesenteric a
4 External iliac a
5 Caudal deep circumflex iliac a
6 Middle sacral a
7. Internal iliac a
8 Ilio-lumbar a
9 Anterior gluteal a
10 Common trunk
11. Ureteral a
12 Deferential a
13 Cranial vesicle a
14 Femoral a

15 Deep femoral a
16 External spermatic (cremasteric) a
17 Pudendo-epigastric trunk
18 Caudal deep epigastric a
19 External pudendal a
20. Caudal superficial epigastric a
21. Urogenital (middle hemorrhoidal) a
22 Hemorrhoidal branch
23 Caudal vesicle a
24 Posterior gluteal a
25 Posterior hemorrhoidal a
26 Perineal a
27 Internal pudendal a
28 Dorsal a of penis

which lies on the dorsal surface of the urethra at the neck of the urinary bladder, and a pars disseminata, which surrounds the pelvic urethra under cover of the urethral muscle Many ducts empty into the urethra dorsally and laterally The epithelium is similar to that of the seminal vesicles The body of the gland is hidden by the seminal vesicles The prostate is quite extensive when the many urethral glands are included with the disseminate portion

The abdominal portion of the ductus deferens is contained in the genital fold It loops over the ureter to lie ventral to the seminal vesicle and opens medial to the duct of the seminal vesicle at the ejaculatory orifice The ejaculatory orifices of both sides are close to the midline on the colliculus seminalis

Although the terminal portion of the wall of the ductus deferens is thickened and somewhat glandular, no distinct ampulla is formed In the genital fold between the ducti deferentes a small uterus masculinus is sometimes present

The testis is supplied by the spermatic artery from the aorta The internal pudendal artery supplies the pelvic genital organs and by way of the ischial arch, the penis The external pudendal artery goes to the preputial area through the inguinal canal

Histology

The epithelium of the epididymis and ductus deferens is pseudostratified columnar in type with stereocilia That of the seminal vesicles and prostate is simple or pseudostratified columnar The mucous secretory cells of the bulbourethral glands are tall The transitional epithelium of the urethra gives way to a stratified squamous type near the external urethral orifice

Semen

The semen is grayish to milky white and contains lumps of gelatin like material The volume is at least 250 cc Ejaculation, therefore, must be prolonged, lasting for

about 8 minutes There are 25 to 50 billion spermatozoa in each ejaculate (see Chapter 3) The gelatinous lumps of the semen come mainly from the bulbourethral gland However, the seminal vesicle fluid seems to enhance the formation of gelatinous material The other accessory glands contribute a less viscous fluid About one fourth of the total volume is contributed by the seminal vesicles, one fifth by the bulbourethral glands, one half by the prostate and urethral glands, and the rest by the testes and epididymes

Spermatozoa are present in the testes by the time the animal is six months of age

FEMALE REPRODUCTIVE ORGANS

Ovaries

The ovaries are suspended by the broad ligament at a position somewhat anterior to the lateral boundary of the pelvic inlet but not close to the kidneys The mesosalpinx is extensive and conceals the ovary There is free communication between the ovarian bursa and the peritoneal cavity ventrally, however The abundance of follicles or corpora lutea makes the size of the ovary difficult to determine A mature ovary containing several large corpora lutea has a very lobate appearance There is a distinct hilus

Fallopian Tubes

The Fallopian tubes are prominent, long (20 cm) and somewhat flexuous The abdominal end is large and possesses fimbria, the uterine end joins the small tip of the uterine cornu

Uterus

The uterus consists of two long (1–1.5 M) flexuous cornua and a short body (5 cm) The relatively short distance between the cervix and the tubal extremities of the uterus makes it necessary that the horns assume a very tortuous course The cervix and vagina are directly continuous, leaving no projection of the cervix into the vagina and no fornix The cervix is long (10 cm) and is distinguished from

the vagina, which is similar in length, by its thicker wall and the many rounded interlocking prominences, which project into the lumen.

The suspensory ligament of the ovary appears as a continuation forward of the broad ligament, which blends with the peritoneum ventral to the kidney. A fold of peritoneum containing a dense cordlike structure represents the round ligament of the uterus. It begins at the anterior extremity of the uterine horn and courses to the inguinal canal where it fades out

Vulva

The vestibular area is long. Ventro-anteriorly the urethra opens into it. The ventral commissure of the vulva is pointed and projects posteriorly. The small clitoris lies in a fossa anterior to the ventral commissure. On each side of the urethral orifice is the opening of the duct of Gartner. There are numerous small isolated vestibular glands. The wall of the urethra contains many cavernous veins. The epithelium is transitional in type.

The ovarian artery on each side is long
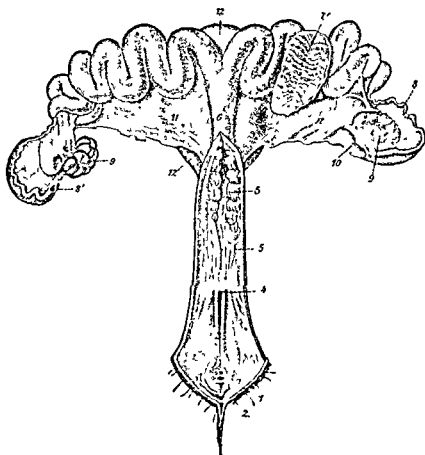
FIG. 1.17—Genital organs of sow; dorsal view. The vulva, vagina, and cervix uteri are slit open. (From Leisering's Atlas)

1. Labium vulvae
2. Glans clitoridis
3. Vulva
4. External urethral orifice
5. Vagina
5'. Cervix uteri
6. Corpus uteri
7. Cornua uteri, one of which is opened at 7' to show folds of mucous membrane
8. Uterine tube
8'. Abdominal opening of tube
9,9. Ovaries
10. Ovarian bursa
11,11. Brood ligaments of uterus
12. Urinary bladder

and tortuous The middle uterine artery arises from the internal iliac artery, with the umbilical branch, and courses in the broad ligament to the cornu of the uterus The vagina and vulva are supplied by internal pudendal branches All anastomose along the genital organs The veins accompany the arteries

Histology

The nonglandular stratified squamous epithelium of the vagina continues forward to include the cervical canal The depth of the epithelium increases considerably, with some cornification, during estrus Leukocytes are abundant in metestrum The vaginal smear, however, is apparently not
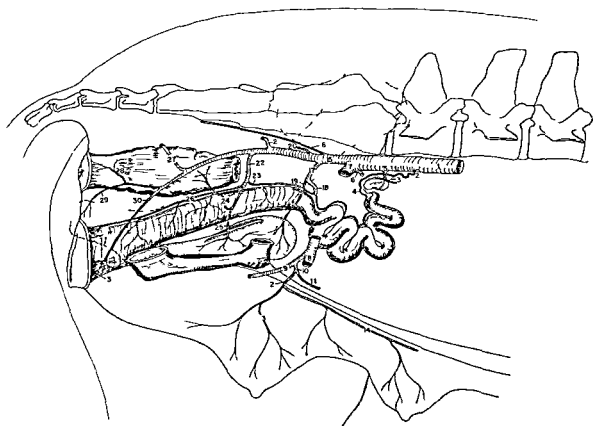
FIG 1 18—Blood supply to female genitalia of sow (From Getty, 1955 Courtesy Burgess Publishing Co and R Getty)

1 Aorta	17 Common trunk
2 Utero ovarian artery	18 M ddle uterine a
3 Ovar an a	19 Cranial ves cle a
4 Anterior uterine a	20 Il o lumbar a
5 Poster or mesenteric a	21 Anterior gluteal a
6 Caudal deep circumflex il ac a	22 Urogenital (m ddle hemorrhoidal) a
7 External iliac a	23 Posterior uter ne a
8 Femoral a	24 Hemorrho dal branch
9 Deep femoral a	25 Caudal vesicle a
10 Pudendal epigastric trunk	26 Vag nal a
11 Caudal deep ep gastric a	27 Posterior gluteal a
12 External pudendal a	28 Posterior hemorrhoidal a
13 Mammary branch	29 Perineal a
14 Caudal superficial ep gastric a	30 Internal pudendal a
15 Internal iliac a	31 A of cl toris
16 M ddle sacral a	

a good indicator of the reproductive state

The epithelium of the uterus is simple columnar Cilia are present in the gland crypts The epithelium may become tall and pseudostratified at proestrum and estrus Soon vacuolar degeneration takes place and the epithelium returns to a low columnar type The labia of the vulva swell and the vestibula area is reddened during early estrus They become flabby and are covered by mucus in late estrus

The epithelium of the oviduct is simple columnar or pseudostratified columnar Some of the cells are ciliated Those that are ciliated become very tall during estrus

Estrus Cycle

The estrus cycle of about 21 days occurs throughout the year Estrus lasts for 2 to 3 days It does not occur during lactation but appears one week after weaning (see Chapter 3) Ovulation is spontaneous, and occurs about 36 hours after the onset of estrus Many ova are cast from each ovary at this time They reach the uterus in 3 days Fertilization takes place in the oviduct Spermatozoa have reached the oviduct, by their own initiative, 7½ hours after copulation They have been known to be propelled to the oviduct by uterine contractions in a matter of a few minutes The semen is probably ejaculated into the body of the uterus because of the nature of the cervix

Placenta

The placenta is diffuse in distribution and is epitheliochorial in regard to layers of contact Circular folds containing secondary ridges are distributed over the chorion except at the cornual extremities Uterine glandular secretions (uterine milk) raise the chorion off the endometrium in spots forming areolae The areolar villi are highly developed The allantois is extensive The embryos are evenly spaced in both cornua even though more ova may have come from one ovary

Although true hermaphroditism wherein the gonads of both sexes are present in the

same animal, is rare, pseudohermaphroditism is rather common in this species Females are more commonly affected than males

ENDOCRINE GLANDS

Pituitary Gland

The pituitary gland lies in the upper part of the pituitary fossa A sheath of dura mater invests it and is fused with its capsule except dorsally where the dural diaphragm does not cover the gland The posterior projecting neurohypophysis (pars nervosa) is continuous with the diencephalon by a slender stalk (infundibulum) The third ventricle extends into the stalk The adenohypophysis includes the pars distalis, pars intermedia, and pars tuberalis The pars intermedia is attached as a narrow rim to the ventral and lateral
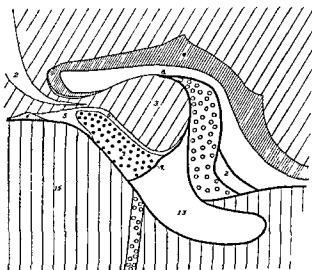
FIG 1 19—Schema of sagittal section through the pituitary gland

1 Brain
2 Third ventricle
3 Pars nervosa
4 Subarachnoid space
5 Pars tuberalis
6 Pars intermedia
7 Pars distalis
8 Anterior limit of the dural diaphragm
9 Dura mater attached to the gland
10 Dorsum sellae (cartilaginous)
11 Dorsum sellae (osseous)
12 Blood sinus
13 Blood sinus
14 Cartilaginous union between presphenoid and postsphenoid
15 Sphenoid bone

surfaces of the pars nervosa. The pars tuberalis is associated with the stalk. The main portion is the pars distalis, which surrounds the pars nervosa except dorsally. The pars nervosa and the pars intermedia constitute the posterior lobe. The pars distalis makes up the anterior lobe. The two lobes are usually separated by a cleft which is a remnant of the cavity of embryonic buccal evagination.

The area between the pituitary dura and the periosteum in the floor of the fossa forms a blood sinus (cavernous). The internal carotid artery, which appears lateral to the stalk, traverses the sinus where it forms a delicate network (rete mirabile). As the internal carotid artery rises to form the arterial circle (circulus arteriosus of Willis) for the brain, it gives off several small superior hypophyseal arteries. The veins go directly into the dural sinus especially at the posterior pole. Portal veins course in the walls of the infundibular stalk from the diencephalon to join sinusoids in the anterior lobe. A vascular connection with the hypothalamus is thus afforded.

The anterior lobe is the largest division of the gland, making up about 60 per cent of its volume. The neural portion of the posterior lobe occupies 25 per cent of the gland. The rest is assigned to the intermedia, tuberalis, and the infundibulum. The pituitary of a 200-lb. pig weighs about .250 gm.

The anterior lobe, which is the portion producing the hormones which make the pituitary the "master gland," contains cells arranged in closely packed groups separated by connective tissue septa. Many blood sinusoids and colloid accumulations are present. There are three basic cell types according to staining properties: acidophils, basophils, and chromophobes. The central areas are basophil-rich and acidophil-poor. The acidophils are found more in the lateral and distal portions. The chromophobes are evenly distributed. In mature animals about one-third of the cells are chromophobes, one-half acidophils, and the rest basophils. The ratio of chromophobes to acidophils is reversed in

baby pigs. The basophils are always the least numerous.

Adrenal Glands

Each gland is long and cigar-shaped, lying medial to the portion of the kidney anterior to the hilus. It is dark reddish-brown. The right gland is attached firmly to the wall of the posterior vena cava. Where the posterior extremity contacts the renal vein, one or more veins open from the gland. Veins may pass into the dorsal abdominal vein and on the right side they may go directly into the posterior vena cava. Small arteries which enter at the periphery may arise directly from the aorta, or from the dorsal abdominal artery, or even from a lumbar artery. The splanchnic nerves enter the adrenal on its lateral surface or pass by it to go to the anterior mesenteric ganglion. The cortex consists, from without inward, of glomerular, fascicular, and reticular zones. Each adrenal weighs about 5 gm. and is about 10 cm. in length in a 200-lb. pig.

Thyroid Gland

The thyroid gland lies in the midline ventral to the trachea near the thoracic inlet; thus it is not related to the larynx. It is dark, narrower from side to side than vertically, and grooved longitudinally on its dorsal surface. It may be 5 cm. long and weigh about 5 gm. in the adult. The posterior extremity is more blunt than the anterior. The blood supply and venous drainage are at the posterior extremity. The one or more arteries usually stem from the right omocervical artery. The vein empties into the anterior vena cava. The simple epithelium of the follicles ranges from low to high cuboidal depending on the state of activity.

Thymus

The thymus occupies the area along the common carotid artery on each side of the neck. Posteriorly the two parts are together, where they lie in the mediastinum in contact with the pericardium. The anterior limit is the origin of the digastri-

cus muscle. The omohyoideus muscle crosses the superficial face of the gland a short distance from its anterior extremity. The thymus is light in color, soft in consistency, and very lobular. It decreases in size in old animals, leaving only a framework of connective tissue. The blood supply arises from available vessels throughout its length.

Parathyroid Gland

The one gland which is present on each side is not in contact with the thryoid but is located in the portion of the thymus which is anterior to the omohyoideus muscle. It is not larger than a small pea (0.05 gm) even in a large animal. It is darker and firmer than the surrounding thymus tissue.

Pineal Gland

The pineal gland is in the form of a tall, narrow cone projecting upward and backward in the midline from the posterior portion of the roof of the third ventricle.

MAMMARY GLANDS

There are two parallel rows of glands extending from the pectoral to the inguinal areas. About six pairs of glands are usually present. The number may vary and there may be more on one side than on the other. Each gland has a teat which has an anterior and a posterior duct. The teats, or nipples, are present in the male but are rudimentary.

The lymph from the superficial areas of the first two pairs of glands drains forward to the posterior cervical lymph nodes. The deep drainage from those glands may follow the blood vessels deeply to the anterior mediastinal nodes. That from the other glands drains into the superficial inguinal (supramammary) nodes.

The glands of the inguinal and abdominal regions are supplied by the external pudendal artery. Perforating branches

FIG. 1.20—Ventral view of cervical region.

A. Mandibular salivary gland
B. Digastricus muscle
C. Thymus
D. External jugular vein
E. Sternocephalicus muscle
F. Outline of thyroid gland
G. Stylohyoideus muscle
H. Omohyoideus muscle
I. Parathyroid gland
J. Mylohyoideus muscle
K. Hypoglossal nerve
L. Common carotid artery
M. Area of larynx
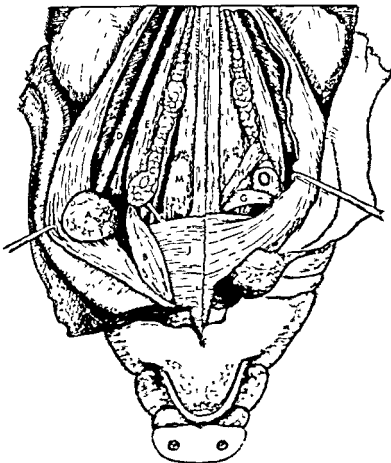
from the internal thoracic artery and per haps branches from the external thoracic artery supply the glands of the pectoral area

The external pudendal and internal thoracic veins are continuous on the deep surface of the gland chain and provide drainage both anteriorly and posteriorly

LYMPHATIC SYSTEM

The thoracic duct begins on the right side of the aorta at the diaphragm as a dilated portion the cysterna chyli, which *receives the lumbar and intestinal trunks* It passes forward across the left side of the trachea and esophagus to empty into the left external jugular vein near its termi nation It may be double anteriorly but is single at its actual termination

The mandibular, parotid, and supra pharyngeal nodes have been referred to in meat inspection as the "cervical glands '

The mandibular lymph nodes are *located near the insertion of the sterno* hyoideus and the anterior border of the mandibular salivary gland There are usually two on each side Afferents are re ceived from the structures of the anterior part of the oral and nasal cavities Effer ents go to the middle and posterior cervi cal nodes

The parotid lymph nodes are reddish *brown and form a chain along the ante* rior border of the parotid salivary gland The afferent vessels come from the area of the eye, ear, and face Efferent vessels go to the suprapharyngeal nodes

The suprapharyngeal nodes are situated on the dorsolateral wall of the pharynx There are usually two on each side, and they are smaller than the mandibular nodes The afferent vessels come from the parotid nodes, the posterior portions of the nasal and oral cavities, and the pharynx Efferents form the tracheal ducts

The middle cervical lymph nodes form a group along the external jugular vein Afferents are received from the larynx esophagus, trachea, and thymus Efferents pass to the posterior cervical nodes

The prescapular or posterior superficial

cervical lymph nodes form a large chain just above the point of the shoulder under cover of the trapezius and omotransver sarius Afferents come from the neck shoulder, and lateral portions of the fore limb Efferents travel to the posterior cervical nodes or the tracheal ducts or the thoracic duct

The posterior cervical or prepectoral lymph nodes are situated at the entrance to the thorax ventrolateral to the trachea Most of the lymph from the head, neck forelimb and thoracic wall drains into or *through these nodes Efferents go into the* tracheal ducts or into the terminal part of the thoracic duct

The lymph from the head drains through the cervical chain of nodes, which also includes the posterior superficial cervi cal nodes, to the thoracic duct or posterior cervical nodes or by way of the tracheal ducts, which run on each side of the tracheal area from the suprapharyngeal nodes to the thoracic duct The right tracheal duct may empty into the external jugular vein independently near the an terior vena cava or may form a short duct in common with the right prescapular ef ferents, the right lymphatic duct

The popliteal lymph nodes constitute a small subcutaneous group posterior to the stifle They drain the lower portion of the hind limb Efferents go to the ischiatic and internal iliac nodes

The superficial inguinal lymph nodes (supramammary in the female) form a large group external to the external in guinal ring They drain the more poste rior mammary glands, the preputial and scrotal areas, the glans penis, and the medial surface of the thigh and leg Effer ents go to the internal iliac nodes

The prefemoral lymph node is a single, elongate body which lies at the anterior border of the m tensor fasciae latae Affer ents are received from abdominal wall and anterior superficial areas of the hip, thigh and leg Efferents go to the external and internal iliac nodes

The small ischiatic lymph node lies near the lesser sciatic notch Afferents are re

ceived from the surrounding area and from some of the popliteal nodes. Efferents go to the internal iliac nodes.

The several small external iliac lymph nodes are associated with the circumflex iliac vessels near the tuber coxae. They receive afferents from the adjacent areas and the prefemoral nodes. Efferents travel to the internal iliac and lumbar nodes.

The internal iliac lymph nodes are a large group near the termination of the aorta. Those of the group which are along the origin of the external iliac artery represent the deep inguinal nodes. They are the principal recipients of lymph from the pelvic organs and limbs and from the nodes of those areas. Their efferents form the lumbar trunks and nodes.

Lumbar lymph nodes are scattered along the lumbar trunks.

The kidneys, stomach, liver, and spleen have, associated with their blood supplies, nodes which drain into the cysterna chyli.

Mesenteric nodes form a chain in the mesentery between the small intestine and the colon. Nodes are also associated with the coils of the large intestine. They all drain into the cysterna chyli.

The thoracic lymph nodes consist of mediastinal, bronchial, and sternal groups

The mediastinal nodes are situated along the trachea at the thoracic inlet and along the aorta. They drain the thoracic viscera, the bronchial nodes, and the diaphragm, and even receive vessels from the liver and spleen. Efferents go to the terminal lymph vessels at the thoracic inlet.

The well-developed bronchial lymph nodes are located at the bifurcation of the trachea and at the right apical bronchus Afferents are received from the lungs, heart, and esophagus. Efferents go to the anterior mediastinal nodes

A single sternal lymph node is situated on the dorsal surface of the more anterior part of the sternum. Afferents come from the ventral thoracic wall. Efferents course with the mediastinal lymph vessels.

The germinal center is located more toward the center of the node than in other
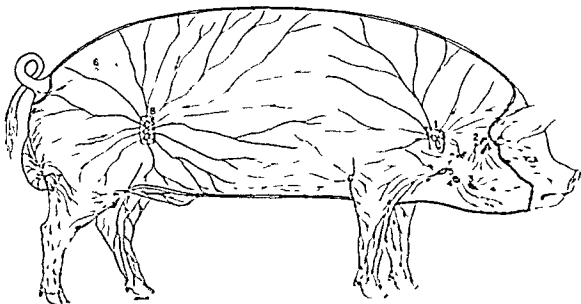
FIG 1.21—Superficial lymph nodes of hog. (From Getty, 1955. Courtesy Burgess Publishing Co. and R. Getty.)

1 Prescapular	5 Prepectoral (posterior cervical)
2 Parotid	6. Sacral
3 Mandibular	7. Popliteal
4 Middle cervical	8 Prefemoral
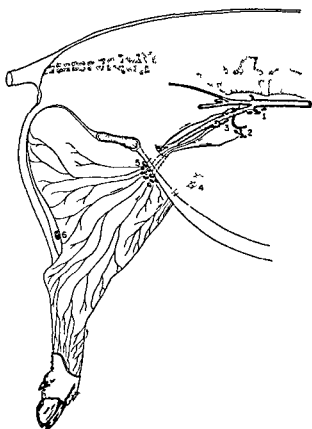
FIG 1 22—Lymph nodes of abdomen and pelvis of hog (From Getty, 1955 Courtesy Burgess Publishing Co and R Getty)

1 Internal iliac   4 Prefemoral
2 External iliac   5 Superficial inguinal
3 Deep inguinal   6 Popliteal

species The peripheral zone is comparable to the medulla of other animals Afferent vessels penetrate the capsule at only a few points, whereas the efferent vessels may be multiple

The mesenteric lymph vessels contain chyle and are called lacteals

Subepithelial lymph nodules appear in the digestive, respiratory, and urogenital systems They are especially abundant in the digestive tract The palatine tonsils are in the soft palate Tubal and paraepiglottal tonsils are also present

MUSCULATURE

The musculature presents no special features However, the muscles which bound the inguinal canal deserve attention

Inguinal Canal

The external inguinal ring is at the lateral border of the rectus abdominis

muscle just anterior to the pubis The ring is in the form of a slit approximately 4 cm in length in the aponeurosis of the external abdominal oblique muscle It is directed downward, outward, and forward The internal inguinal ring is the area bounded anteriorly by the last muscle fibers of the internal abdominal oblique and posteriorly by the external abdominal oblique aponeurosis The direction of the ring is upward, outward, and forward The two rings are about 7 cm apart anteriorly but close together posteriorly The canal, which is the area interval between the two rings, is deep to the external oblique aponeurosis and lateral to the rectus abdominis The canal is much longer anteriorly than posteriorly and its direction is forward and outward Through the center it is about 5 cm in length The spermatic cord, enveloped in tunica vaginalis and cremaster muscle, passes through the more anterior portion of the canal The cremaster, a large slip from the internal oblique muscle, passes at least part way through the canal in the female and may be associated with the termination of the round ligament of the uterus No peritoneal evagination occurs, however In both sexes the external pudendal vessels pass through the posterior, short, portion of the canal Hernia can result from tendency of the rings to become superimposed

CIRCULATORY SYSTEM

The relatively small heart lies along the sternum in a less upright position than is usually the case The fibrous pericardium is attached to the sternum from the second sternabra to the xiphoid cartilage at the diaphragm It pushes the mediastinal pleura against the costal pleura at the cardiac notches The apex of the heart is in the midline just anterior to the diaphragm The heart is turned so that the sternal surface is contributed by the right ventricle, except at the apex which is formed by the left ventricle The right ventricle has a large moderator band

The brachiocephalic and left subclavian arteries come off in turn from the aorta The common carotid arteries usually arise

by a very short trunk from the brachio cephalic artery The costocervical, deep cervical, and vertebral arteries usually arise together However, on the left side they may be separate The bronchial and esophageal arteries are usually separate The abdominal portion of the phrenico abdominal artery may arise from the aorta near the renal artery independent of the phrenic portion

The intercostal veins drain into the vena

hemiazygos which runs along the left side of the vertebral bodies to empty with the left coronary vein into the right atrium below the opening of the posterior vena cava The right and left axillary and ex ternal jugular veins unite at the thoracic inlet to form the anterior vena cava This confluence may be joined also by larger than usual internal jugular veins The anterior vena cava is ventral to the brachio cephalic artery
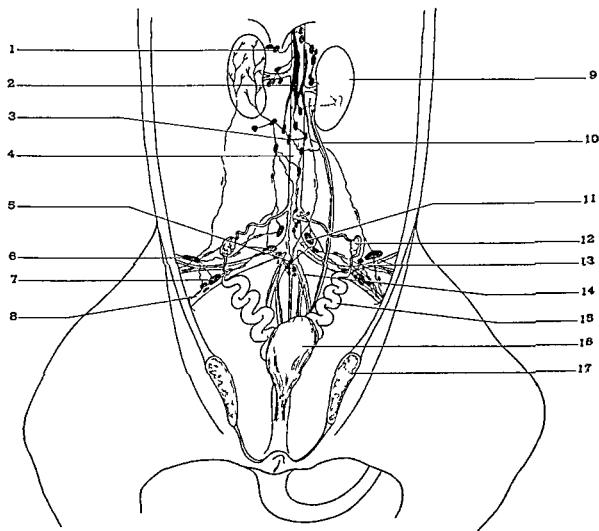
FIG 1 23—Lymphatic drainage of abdomen and pelvis of sow (From Getty, 1955 Courtesy Burgess Publishing Co and R Getty)

1 Adrenal lymph nodes	10 Ureter
2 Cisterna chyli	11 External iliac artery
3 Aortic lumbar nodes	12 Ovary
4 Aorta	13 Circumflex iliac artery
5 Internal iliac nodes	14 Internal iliac artery
6 External iliac nodes	15 Uterus
7 Deep inguinal nodes	16 Bladder
8 Femoral artery	17 Ilum
9 Kidney	

FIG 1 24—A dissection of the right side of the pig (From Foust and Getty, Iowa State University Press, 1954)

I, IV, XII. Costae

1 Ala ossis sacrum facies articularis
2 Dotted line indicates outline of Os coxae
3 Os pubis
4 Section of Os ischii (3 and 4 are united by medial border of Foramen obturatum)
5 M semimembranaceus
6 M semitendinosus
7 M biceps femoris
8 M gluteus
9 M retractor penis
10 M longissimus dorsi et cervicis
11 M ilio psoas
12 M obturator internus
13 M pectineus
14 Aponeurosis of M gracilis et M adductor
15 M ischiocavernosus
16 M bulbocavernosus
17 Lig retractoriductum (cut edge)
18 Fascia glutaea
19 M obliquus abdominis externus tendo membranaceus
20 M obliquus abdominis externus tendo membranaceus
21 Part of M of liquus abdominis externus raised to show relationship of M obliquus abdominis internus and M rectus abdominis
22 M of liquus abdominis internus
23 M obliquus abdominis internus tendo membranaceus
24 M transversus abdominis
25 Muscular part of M transversus abdominis showing thin aponeurosis of M obliquus abdominis internus covering it
26 Arcus costarum
27 M retractor costae
28 Fascia transversalis
29 M serratus dorsalis caudalis
30 Fascia lumbodorsalis
31 Diaphragma
32 M latus mus dorsi
33 M serratus dorsalis cranialis
34 Ligamentum dorsoscapulare

35 M trapezius
36 M rhomboides cervicalis et capitis
37 M splenius
38 M serratus ventralis cervicis
39 M brachiocephalicus
40 M cutaneus colli
41 M omotransversarius
42 M scalenus
43 M pectoralis minor, pars scapularis
44 M pectoralis minor pars humeralis
45 M pectoralis major pars descendens
46 M pectoralis major, pars transversa
47 M cutaneus trunci
48 M rectus abdominis covered by tendo membranaceus
49 M praeputiales cranialis
50 M praeputiales caudalis
51 Ostium praeputiale
52 Orificium diverticuli praeputialis
53 Cut border of Praeputium
54 Outline of Diverticulum praeputiale
55 Penis
56 Panniculus adiposus
57 Fascia
58 Fascia inguinalis
59 Tunica dartos
60 Cut edge of Tunica vaginalis testis et funiculi spermatici
61 Vascular plexus (62 63 64 are covered by Tunica vaginalis et testis funiculi spermatici)
62 Testis
63 Epididymis
64 Cut edge of Tunica dartos continuous with Septum scroti
65 M cremaster externus
66 Annulus inguinalis subcutaneus in M obliquus abdomin externus
67 Peritonaeum
68 Glandula bulbourethralis
69 Prostata
70 Glandula testiculosa
71 A et V iliaca interna

72 A et V glutaea caudalis
73 A et V pudendalis interna
74 Artery and vein supply Glandula bulbourethralis
75 A et V umbilicalis
76 A et V glutaea cranialis
77 A et V femoralis
78 A et V circumflexa ilium profund Ramus caudalis
79 A et V epigastrica profunda caud
80 V epigastrica profunda cranialis
81 A et V thoracica interna
82 A et V brachialis
83 Plexus brachialis
84 N phrenicus
85 N cutaneus femoris lateralis
86 N femoralis
87 N obturatorius
88 Nerve to Prostata and Glandula vesiculosa
89 N ischiadicus
90 N pudendalis
91 Lymphonodi with A femoralis
92 N subhilaeus
93 Lnn inguinales superficiales
94 Lnn cervicales superficiales
95 Lnn paroticula
96 Lnn cervicales medii
97 Thymus
98 Pericardium covered by pleura
99 Pulmo, lobus apicalis
100 Pulmo, lobus cardiacus
101 Pulmo lobus diaphragmaticus
102 Peritonaeum parietale
103 Intestinum tenue
104 Omentum majus
105 Peritonaeum pelvis
106 Wall of Intestinum rectum
107 Ureter
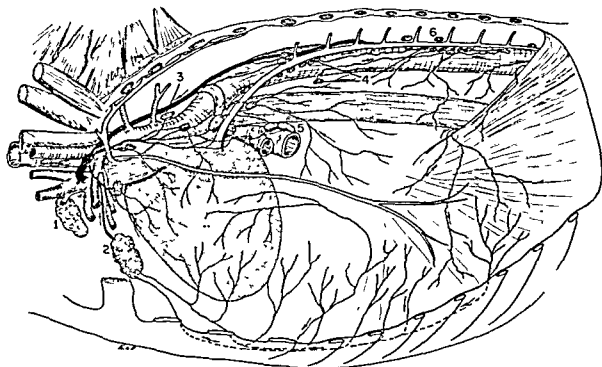
FIG 1.25—Deep lymph nodes of pig. (From Getty, 1955. Courtesy Burgess Publishing Co. and R. Getty.)

1 Prepectoral	4. Caudal (aortic) mediastinal
2 Sternal	5. Bronchial
3. Cranial mediastinal	6. Intercostal

The anterior vena cava is used as a site for collecting blood or administering various agents in young pigs. The needle passes just anterior to the tip of the sternum in a posterodorsal direction. In larger animals the external jugular vein may be reached in the depression between the point of the shoulder and the ventral neck muscles. The posterior auricular vein, which follows subcutaneously the posterior border of the ear on the convex side, is usually accessible for injections.

NERVOUS SYSTEM

The brain is relatively small and deeply seated in the skull. The spinal cord ends at the midsacral level. There are eight pairs of cervical nerves. The others correspond to the number of vertebrae in each region, except in the coccygeal area. The nervous system in general presents no special features.

SKIN AND SENSE ORGANS

The skin, which is medium in thickness, is thickest on the ventral aspect of the neck. The bristles, which are sparsely placed leaving some areas bare, are long and coarse on the back and neck. They occur in groups of three. The subcutis contains a thick panniculus adiposus. Sebaceous glands which open into the hair follicles are small and few in number. Large ones are present at the entrance of the preputial diverticulum, where coiled tubular glands are also found. Special coiled tubular glands occur on the medial side of the carpus, where there are cutaneous invaginations, and on the digits and interdigital spaces. Compound tubular glands are present in the skin of the snout. Ordinary sweat glands are not present.

Each digit has a well-defined hoof, or claw. The horny bulbs continue downward, limiting the sole to be a small area close to the wall.

In addition to the mucous gland which is adjacent to the cartilage of the third eyelid, at a deeper level there is a large, distinct mixed gland. The lacrimal gland is of the mucous type. The two lacrimal ducts do not form a lacrimal sac but pass

FIG 1 26—Three quarter view
of ventral part of neck

1   Anterior tip of sternum
2   First rib
3   Confluence of veins to form
    anterior vena cava
4   Axillary vein
5   External jugular vein
6   Thyroid gland

into separate openings into the nasolacrimal duct, which empties into the posterior part of the ventral meatus The tarsal glands are small No lashes are found on the lower eyelids The tapetum of the choroid membrane is not present The pupil is oval

The osseous external acoustic canal is very long straight and directed ventromedially The tympanic membrane is circular and located deeply The opening of the auditory tube is in the upper wall of the pharynx close to the posterior naris

REFERENCES

ASDELL S A 1946 Patterns of Mammalian Reproduction Comstock Publishing Co Ithaca N Y

BISWAL G MORRILL C C AND DORSTEWITZ E L 1954 Glands in the submucosa of the porcine colon Cornell Vet 44 93

CARL, B N AND DEWHIRST W H 1942 A method of bleeding swine Jour Amer Vet Med Assn 101 495

ELLENBERGER W AND BAUM H 1943 Handbuch der vergleichenden Anatomie der Haustiere Springer Verlag Berlin

FOUST H L AND GETTY R 1954 Anatomy of Domestic Animals 3rd ed Iowa State College Press Ames Iowa

GETTY R 1955 Atlas for Applied Anatomy Burgess Publishing Co Minneapolis

HABEL, R E AND BIBERSTEIN E L 1957 Fundamentals of the Histology of Domestic Animals Comstock Publishing Co Ithaca N Y

MCKENZIE F F MILLER J C AND BALGNESS I C 1938 The reproductive organs and semen of the boar Univ Mo Res Bull 279

PATTEN B M 1959 Embryology of the Pig The Blakiston Co New York

SISSON S AND GROSSMAN J D 1953 Anatomy of Domestic Animals W B Saunders Co Philadelphia

ST CLAIR L E 1943 Hypophysectomy and its physiologic effects in the pig (Sus scrofa domestica) Iowa State Coll Jour Sci 20 16

TURNER C W 1952 The Mammary Gland Lucas Brothers Columbia Mo

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CHAPTER 2

Hematology and Hematopoietic Organs*

BLOOD

Introduction

Data on swine hematology are scattered, conflicting, and some are of questionable validity. Good literature reviews are available in the accounts of Scarborough (1931-32), Kernkamp (1932), Oglesby *et al.* (1931-32), Gardner (1947), Wirth (1950), and Seamer (1956).

It is a well-established fact that various physiological and nutritional states affect the hematological picture in all animals. Furthermore, the works of Kernkamp (1932), Venn (1944), and Gardiner *et al.* (1953) have shown that varied environmental conditions also influence the hematologic state of swine. Swenson *et al.* (1955) have demonstrated that the ration of the sow significantly affects the hematology of the newborn pig. Kernkamp (1932) proposed a set of approximately normal total blood cell count values for pigs from birth to 3 months, giving separate values for those reared on concrete and those having access to the soil. Since swine herds exist under such different physiological, nutritional, and environmental conditions, the results of various investi-

gators show considerable diversity (Table 2.1).

Blood groups. Compared with other domestic animals, very little work has been done on the blood groups in swine. Most investigations of cellular antigens have been done in regard to studies of hemolytic disease of newborn pigs.

Szent-Ivanyi and Szabo (1951) studied blood groups in 1,120 pigs by the chessboard method and in 450 pigs with the cross-adsorption method. They reported four antigens (A, B, C, D) in the erythrocyte of the pig, and four iso-antibodies against them (anti-A, anti-B, anti-C, and anti-D) in the serum. These workers found that in the agglutination tests and the adsorption tests, antigen A was the most active. With cross-adsorption methods it was possible to demonstrate a close relationship between the A erythrocytes in the pig and in man.

A considerable number of cases of unexplained anemia in newborn pigs have been reported by many workers. Bruner *et al.* (1949) found that certain antibodies in the colostrum of the sow resulted in anemia and death of newborn pigs after suckling. These authors believed that sows became sensitized with commercially prepared hog cholera virus. Doll and Brown (1954) reported a case of hemolytic disease of the newborn pig and suggested that the sow was sensitized following administ-

TABLE 21

Hemograms Reported on Swine

Author	No.	Age and/or Weight	Hemoglobin (Gm/100 cc)	RBC (millions/cu mm)	RBC Size (μ)	WBC (thousands/cu mm)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Eosinophils (%)	Basophils (%)
Culver, 1927	24	4 mo	13.0	8.4		19.0	37.0	51.6	4.6	5.2	1.7
King and Wilson, 1910	43		12.5	6.4		19.9	35.8	54.2	3.7	4.5	1.7
Palmer, 1917a	25	2.5 18 lbs	8.6	3.8		13.5	32 13	63 25	2 63	1 28	0
	25	100 lbs	11.8	6.2		18.3	39 79	52 21	79	3 42	25
Venable, 1910	50			2 8-9 6		11 3-20 0	26 0	57 0	2 2-5 2	0 7-8	0 7-1 5
Kobayasha, 1924	12		12 13 2	6 5	5 3	20 6 0	22 0	23 7	2 7	0 1	0 1
Seelhorst et al, 1931-32	215		5 9 17	5 0	6 1	8 0 20 4	30 5	52 5	3 3	4 5	0 1
Oshdy et al, 1931 32	51		11 9±0 23	7 9±0 1		21 5 = 662	36 1 = 1	58-76 0	0 21 5	0-15 4	0-2 4
							41 2±1 035	141 2±1 035	1±0 4	1 1±0 3	0 4±0 06
								lg 2±1 0			
								11 2±0 85			
Craft and Moe, 1932	217	birth	9 7		4-7	10-30	54 6	14 8		0 1	
Itazoy, 1938		180 d	12 6		3-7	5-20	30 6	64 9		4 4	
		birth-3 da			5-8	10-30	40-90	10-50	2-5	0-10	0-2
		1 wk-1 mo			5-10	10-30	20-60	30-70	5-10	0-5	0-1
		2-7 mo				10-30	30-70	40-60	5-10	3-10	0-1
		1-2 yr					20-50	20-50	2-10	2-5	0-1
Venn, 1944	18	5-84 da	12 1±1 3	5 6±0 7		5 0-20 1	25 0-62 1	34 4-70 8	1 9-4 0	0 43-2 9	0-18
Ohta, 1949	6		14 2	6 6		17 1	10 2	50 4	1 7	5 9	1 6
Wurth, 1959	41	5-36 mo		5 8	6-6 2	15-20	58 0	36 8	1 9	2 3	0 3
Wintrobe, 1951	390		15 0	7 9	5 5	7 0-20 0	32-78 7	18-61 7	0 3 4 0	0-3-10 3	0 1
Payne, 1952	51	1 da	12(0-17)	7 (5 9)		15 (7-20)	40 0	50 0	8 0	2 0	<1
Gardner et al, 1953	44	8 da	10(9-17)			4-19 0	71 1	26 5			
	44	15 da	7 5			6-23 0	41 1	56 5			
			8 2			5-22 0	32 3	65 5			

TABLE 2 1 (continued)

HEMOGRAMS REPORTED ON SWINE

Author	No	Age and/or Weight	Hemoglobin (Gm/100 cc)	RBC (millions/cu mm)	RBC Size (μ)	WBC (thousands/cu mm)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Eosinophils (%)	Basophils (%)
Luke, 1953b	30	adult sows	11 2±1 6 5	5 1±1 13		10 0-25 5 — 15 9	35 0	63 0		2 0	
Ross, 1953	30	bacon pigs				13 7	28 0	71 0		1 0	
	50	8-16 wks A M				30 6	36 3	62 0		1 6	
	20	8-16 wks P M				19 1	25 4	72 7		1 9	
	12	adult ♂ cast					52 94	44 36	1 34	1 12	0 14
	12	adult ♀ cast					50 7	46 46	1 45	1 06	0 13
	6	adult boars					44 36	48 90	1 53	4 93	0 26
	6	adult sows					30 45	62 70	1 00	5 57	0 20
Svenson et al, 1955 Dukes, 1955 Holman, 1956 Köhler, 1956	43	adult sows	11 95	7 4		7 4±1 8 17 1 14 7±4 5	41 0 53 0=11 (ind cos & bas)	47 0 38 0=13 0	8 0 0 4 0=2 0 4	2 5 0±2 0	<1
		newborn	12 0	5 6±0 7	6 0	6 0	60 (incl eos & bas)	30 0-35 0	scanty	0	

tration of crystal-violet hog cholera vaccine, since the vaccine was prepared from the blood of pigs bled at the height of the virus infection. Goodwin and Saison (1956) reported a breed difference in the iso-antibody response after vaccination with crystal-violet swine fever vaccine. They vaccinated 97 Essex/Wessex sows with crystal-violet vaccine and found that iso-antibodies were almost always present and often in high titer. However, in 69 Large White sows, iso-antibodies were absent in two-thirds of those tested.

In experiments by Goodwin and Coombs (1956), the relationship of the A antigen-antibody system of the pig and hemolytic disease of the newborn was studied. They reported that, since the A antigen is absent from the red cells of newborn group A pigs and appears with increasing concentration during the first few months of life, it is consistent with the possibility that anti-A could produce hemolytic anemia in the newborn. It is apparent that more research concerning blood groups in swine is needed before their role in the etiology of swine diseases is determined.

Technics. Several standard reference texts are available and should be referred to for the technics of procuring and handling blood, total counts, differentials, stains, identification, and other blood determinations: Osgood and Ashworth (1937), Osgood (1940), Wintrobe (1951), Undritz (1952), Boddie (1956), Diggs et al. (1956), and Gradwohl (1956).

Site of obtaining blood. According to Gardner (1947) and Boddie (1956), pig blood may be procured by snipping the tail or bleeding the ear. Most venapunctures in pigs are obtained from the vena cava, but Staub (1954) considered this too dangerous for piglets and proposed using the cephalic vein instead. Carle and Dewhirst (1942) worked out a satisfactory technic for withdrawing blood from the anterior vena cava.

Anticoagulants. Lewis and Shope (1929) used 2 mg. of powdered potassium

oxalate per ml. of blood. While Bunce (1954) found oxalate satisfactory for other farm animals, he preferred 1 mg. of sodium citrate per ml. of blood for use with pig blood. According to Hewitt (1932), it was necessary to use 60 mg. of sodium citrate per 10 ml. of blood to prevent coagulation. Kernkamp (1933) observed that oxalated swine blood showed an increase in the relative per cent of lymphocytes and a decrease in the relative per cent of neutrophils as well as a decrease in total number of red and white blood cells. This became more apparent as the interval between the time of drawing and the time of counting increased.

Blood Values

Blood volume. Hansard et al. (1951, 1953) determined the blood volume of swine weighing from 10 to 675 lbs. to be 7.4 to 3.4 ml. per 100 gm. of body weight. They observed a progressive decrease in total blood volume per unit of body weight with growth and attributed this change to excessive fat in the maturing pig. According to these workers, the newly born pig has a high blood volume. Bush et al. (1955b) made 71 blood volume determinations on 31 normal swine ranging in weight from 22 to 242 lbs. Their results agreed favorably with those of Hansard et al. (1951, 1953) though 15 per cent higher. Bush et al. found the red cell volume varied from 3.2 ml. per 100 gm. in a 22-lb. pig to 2.1 ml. per 100 gm. in a 242-lb. pig. The plasma volume in the same animals varied from 6.2 to 3.5 ml. per 100 gm. Hansard (1956) determined the mean total blood volumes for 9 ± 2-, 46 ± 30-, and 620 ± 71-pound swine to be $7.1 \pm .2$, $6.6 \pm .2$, and $3.5 \pm .5$ ml. per 100 gm., respectively.

Coagulation time. Payne (1952) listed an average coagulation time of 4 minutes for swine blood. According to Dukes (1955), Amendt gave the coagulation time at 25°C. of 3½ minutes. Smith (1912), quoting Nasse, gave a ½-1½-minute coagulation time. King and Wilson (1910)

determined a coagulation time of 2 minutes and 23 seconds Using Quick's method, Muhrer *et al* (1942) found an average whole blood coagulation time of 6 2 minutes for 5 normal animals They reported a strain of swine with a defective clotting mechanism In these abnormalities a globulin free fraction prepared from normal blood reduced the coagulation time

Prothrombin time. Muhrer *et al* (1942) reported 9 1 minutes for 5 swine

Bleeding time. Muhrer *et al* (1942) reported 2 9 minutes for 5 swine

Clot retraction time. Muhrer *et al* (1942) reported 68 minutes for 5 swine

Blood sedimentation rate According to Bunce (1954), the blood sedimentation rate is quicker in the pig than in other farm animals He suggested taking the reading at the end of 8 hours His average value at this point was 3 7 mm (3 2–8 0) for 6 pigs

Hemoglobin Albritton (1952) gave the blood hemoglobin value for 1–2 hour pigs 11 8 (11 4–12), 1–10 days 8 1 (5 4–10 1) and adult females 13 8 Gardiner *et al* (1953) found that a postnatal decline in hemoglobin was twice as great in litters on concrete floor as in those on ground and pasture during the first week of life Hematocrit values were correlated with the hemoglobin values Barber (1955) observed a hemoglobin decline in indoor reared pigs which persisted through the seventh week Outdoor raised pigs did not exhibit a similar fall in hemoglobin Swenson *et al* (1957) found a consistent hemoglobin range of 9 2 to 15 3 for Durocs and attributed a range of 8 1 to 12 4 for Humpshires to the maternal ration during gestation Wintrobe (1951) listed the normal hemoglobin value as 15 Wintrobe (1951) reported a *mean corpuscular volume* of 58 cu μ a *mean corpuscular hemoglobin* of 19$_{\mu\mu}$ gm , and a *mean corpuscular hemoglobin concentration* of 33 per

cent for 300 pigs Hemoglobin values by other authors are given in Table 2 1

Specific gravity. According to Albritton (1952), the specific gravity of whole blood of young pigs is 1 046 and that of plasma 1 022 Senftleben (1919) gave an average specific gravity 1 050 (1 042–1 055) and King and Wilson (1910) listed 1 059 as the specific gravity

Blood chemistry. Relatively little chemical research has been conducted on swine blood Available values including the water and solids of the blood plasma proteins amino acids vitamin content hormones, minerals enzymes coenzymes, and electrolytes are given by Albritton (1952) and compared with other farm animals The reader is referred to this monumental work for such data Hewitt (1932) in a study of the blood chemistry of normal and cholera infected swine reviewed the literature up to that time

Erythrocytes

Numbers Various investigations show a wide range in numbers of red blood cells 3 855 000–10 000 000 with most of them 5 to 8 million (Table 2 1) Kohaniwa (1928) gave a leukocyte erythrocyte ratio of 1 319 This was considerably less than for other livestock Senftleben (1919) observed that male piglets had a higher red blood cell count than females This difference disappeared after weaning Doyle *et al* (1928) found an average of 5 200 000 red blood cells in 29 one-day old pigs

Reticulocytes Wirth *et al* (1939) observed 3–4 per cent reticulocytes in normal adults and in piglets 1 1–13 8 per cent Fraser (1938) recorded 2–5 per cent reticulocytes in piglets from birth to 3 days old 2–40 per cent in those 1 week to 1 month of age, and 0 1–1 5 per cent in older pigs

Size According to Fraser (1938), the red blood cells of swine vary more in size than those of any other domestic animal (2 8–10μ) He found that normoblasts and Jolly bodies were usually present in small

numbers Albritton (1952) listed the size of the red blood cell as 6 0μ. Kohanawa (1928) found that 4,896 cells from 6 animals averaged 5 3μ According to Swenson *et al.* (1957) Duroc pigs were born with a considerably smaller erythrocyte (66 5 cu. μ) than Hampshire pigs (90 0 cu. μ) but concluded this difference probably was due to maternal ration rather than breed difference Other data on size are given in Table 2.1.

Inclusions. Dinwiddie (1914) described some small, spherical, ovoid or crescent-shaped bodies in the pig red blood cell Splitter (1953) mentioned these coccoid-like inclusions which he observed in a few apparently normal pigs, as well as in pigs with acute infection with *Eperythrozoon suis* In our own specimens we observed a similar inclusion in about 50 per cent of the animals

Polychromasia. M a n y investigators found polychromasia characteristic of pig blood Wirth (1938) studied the effect of hemorrhage on the blood picture by removing about one half of the blood In the pig he observed no basophilic stippling of the red cells, relatively few nucleated red blood cells and Jolly bodies, but hundreds of thousands of polychromatic erythrocytes

Normoblasts. A few normoblasts are encountered in almost all normal blood smears Regner (1923) counted 6 per 100 and Meyer (1924) 30 per 300 white blood cells

Erythrocyte fragility. Albritton (1952) listed the initial hemolysis of pig red blood cells at 0 74 per cent NaCl solution and

complete hemolysis with 0.45 per cent NaCl. Hudson (1955) compared the erythrocyte fragility of 20 pigs 72 hours of age or under and 20 seven month old pigs and found greater fragility in the erythrocytes of young pigs. He concluded that the hematological picture of baby pigs is in a transitory state.

Packed c e l l volume (hematocrit). Bunce (1954) made a study in six pigs over a period of 15 minutes to one hour varying the r.p.m. from 1,000 to 3,500 A condensation of the original data is shown in Table 2 2.

Albritton (1952) gave a hematocrit value of 41 5 per cent with a range from 30–53 per cent He also listed the hematocrit values for pigs 1 to 12 hours 39 6 per cent (39–40 per cent), 1–10 days 25 0 per cent (18–36 per cent), and adult females 40 8 per cent

Swenson *et al.* (1957) found the average hematocrit value for 35 Durocs 36 hours to 8 weeks of age (29 8–44 4 per cent) slightly higher than those of 12 Hampshires of the same ages (24 9–41 8 per cent). Other hematocrit values given were. Oglesby *et al* (1931–32) 47 8±0 96 per cent, Wintrobe (1951) 46 3 per cent, Payne (1952) 58 per cent and Gardiner *et al.* (1953) I day old 38 1 per cent, 8 days old 30 0 per cent and 15 days old 34 9 per cent. Hematocrit values on 18 three month-old pigs in our own investigations averaged 39 3 per cent (3,000 r p m for 30 minutes).

Life span Bush *et al.* (1955a) using C^{14} labeled glycine found that the mean red cell survival time in growing swine was 62 days Using tracer doses of Fe^{59}, Jensen

TABLE 2 2
PACKED CELL VOLUME, PER CENT

Pig No	R B C	15 Min 1,000–3,500 r p m	60 Min 1,000–3,500 r p m
1	4 1	77 0–47 5	60 0–39 5
2	6 8	87 0–49 0	71 0–42 0
3	6 1	88 0–31 0	38 0–26 0
4	5 9	76 0–35 0	49 0–27 0
5	6 8	94 5–39 5	58 0–27 25
6	6 3	91 5–33 0	64 0–27 5

et al (1956) determined the average apparent red cell life span to be 63 ± 16 days Bush *et al* (1956) measured a life span of 63 days using radioactive chromium in 26 pigs

Fetal blood picture Jones *et al* (1936) used the fetal pig in a study on changes occurring in the blood picture during fetal life Their results are shown in Table 2 3

Wintrobe and Shumacker (1936) studied erythrocytes in fetuses and newborn animals They used the pig as one of the experimental animals and reported the following

It is shown that the red cell count hemo globin and volume of packed red cells are at first very low as compared with those of adults of the same species whereas the red corpuscles themselves are very large chiefly nucleated and contain correspondingly high amounts of hemo globin As the fetus develops the number of red cells amount of hemoglobin and volume of packed red cells increase whereas the mean size of the cells their mean corpuscular hemo globin and the proportion of immature erythro cytes decrease Mean corpuscular hemoglobin concentration however remains essentially con stant throughout

Authors' observations Table 2 4 sum marizes the results of our observations Swine red blood cells are biconcave discs similar to those of other farm animals In our observations on 60 animals under six months of age there appeared to be great variation in cell sizes Polychromasia and poikilocytosis were observed in most of the smears As many as 5 normoblasts were seen in counting 100 white blood cells

Molina and Gonzalez (1940) drew similar conclusions from observing the blood of young pigs These characteristics are less prominent or disappear completely with increasing age The mean color index on 17 three month old pigs was 0 607 Kohler (1906) gave a value of 0 7 for pig blood

Leukocytes

The white blood cells of swine have been described by Giltner (1907) Palmer (1917a) Kohanawa (1928) Oglesby *et al* (1931–32) Kennedy and Climenko (1931) Fraser (1938), and Venn (1911)

Table 2 1 includes the white cell counts recorded by various investigators In re viewing the literature up to that time Scarborough (1931–32) concluded that the total white cell counts were 20–50 per cent higher in young pigs and higher in the males a lymphocytosis accompanied lac tation and a digestive leukocytosis occur red within 3 to 5 hours after feeding It has been well established by Kernkamp (1932) Fraser (1938) Venn (1911) Gardiner *et al* (1953) and Luke (1953a) that the total number of white cells in normal piglets decreases after birth and that an increase takes place at about 2 weeks of age resulting in a lymphocytic blood picture According to Gardiner *et al* (1953) the white blood cells were not affected by variations in environment such as concrete floors vs dirt floors or ground and pasture Wirth *et al* (1939) stated that the numbers of white blood cells in swine are high He gave 10 000–15 000 for young animals and 15 000–20 000 for grow ing animals and for the most part lympho-

TABLE 2 3
FETAL BLOOD PICTURE

	42 Days	107 Days	2–42 Days Perinatal
Total erythrocyte count in millions	0 74	3 0	5 2
Hemoglobin in grams per cent	3 6	6 76	9 7
Mean corpuscular volume in μ^3	216 0	178 0	
Mean corpuscular hemoglobin in gm $\times 10^7$	56 2	21 0	
Average cell diameter in μ	8 7	6 01	
Range of cell diameter in μ	2 0 16 0	4 0 8 5	
Reticulocytes, per cent (1 000 cells counted)	29 8	8 0	

cytic in nature. Swenson *et al.* (1957) found a gradual increase in total leukocytes in Durocs from around 7,000 at birth to 19,000–20,000 at 5 weeks.

Effect of digestion. Regner (1923) studied the effect of digestion on the differential white blood cell count. During digestion lymphocytes decreased 11.2 per cent and neutrophils increased 12.1 per cent. At 2 to 5 hours after eating he found 42.3 per cent lymphocytes and 49.8 per cent neutrophils, but 12 to 17 hours after feeding there were 54.8 per cent lymphocytes and 37.5 per cent neutrophils. Other cells were not affected.

Effect of exercise. Palmer (1917b) found that by exercising 15 normal pigs the total white blood cell count increased and the blood picture changed from lymphocytic to neutrophilic.

Effect of heat. Exposure to the sun caused changes in the blood picture similar to those produced by exercise (Palmer 1917b).

Effect of adrenal hormones. Luke (1953c) observed a lymphopenia and neutrophilia with a sharp increase in the total white cell count within two hours following the administration of adrenocorticotrophic hormone and adrenal cortical extract to swine.

Neutrophils. *Various nuclear shapes such as ring, spiral, U, S, Z, M, 8, or double 8 forms, in addition to the segmented forms, have been described* in neutrophils of the pig. According to Giltner (1907) and Palmer (1917a), the nucleus, if segmented, is extremely polymorphous. Venn (1944) found more than three lobes uncommon. Kennedy and Climenko (1931) attributed a left-handed Arneth count to these young forms. Fraser (1938) found 13 per cent or less of the neutrophils had more than three lobes and observed none with more than five lobes. Fraser (1938) and Venn (1944) gave Schilling hemograms. In pigs up to 12 weeks of age Venn found the stab forms (73 per cent) exceeded the segmented

forms (21 per cent). In young and adult pigs Fraser observed approximately 10 per cent band types. According to Wirth (1938), a certain number of young forms exist in the blood of healthy swine.

Luke (1953a) observed a well-marked lymphopenia and a neutrophilia in the sow at parturition. This may be manifested from 6 to 30 days prior to parturition or may be delayed until farrowing has commenced. According to Luke (1953b), a comparative lymphopenia was present in the newborn pig. He found wide variations in the total white cell counts made at weekly intervals.

According to Kohanawa (1928), their size varied from 6.6 to 15.4μ, most frequently 11.0μ, and the nuclear segments varied from 1 to 6 with 3 segments occurring most usually. Kohanawa found only one segment in 0.9 per cent of the neutrophils. Venn (1944) listed the average size at 11μ. Recent morphological studies of polymorphonuclear blood cells, particularly neutrophils, have revealed the presence of a sex chromatin appendage ("drumstick") in the female of many species. They were first described by Davidson and Smith (1954) in the human species and later in the domestic and laboratory animals by Smith and Calhoun (1956). Fig. 2.1 shows a ring-shaped nucleus with a "drumstick."

In our smears of pig blood variable forms of the nucleus of the neutrophil were evident. All the shapes described above were observed. Fig. 2.1 shows a ring-shaped nucleus. While hypersegmentation of the nucleus is not characteristic of the neutrophil of this species, one with 6 segments is shown in Fig. 2.2.

Lymphocytes. Giltner (1907) and Venn (1944) described two types of lymphocytes, and Palmer (1917a) classified these cells into three groups — small, medium, and large. According to Fraser (1938), the nucleus may be spherical, oval, or kidney-shaped, but a "bilobed form is not seen in the pig." Both these investigators found azurophile granules rarely and Fraser ob-

TABLE 24

HEMOGRAMS ON SWINE USED IN THIS STUDY*

No. of Animals	Age	Sex†	Breed	Hemoglobin (Gm/100 cc)	R B C (millions/cu mm)	W B C (thousands/cu mm)	Band Neutrophils (%)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Eosinophils (%)	Basophils (%)
5	21-25 days	♂	Durocs and Duroc Chester White cross	12.7	5.1	10,710	9.4	31.8	45.4	8.2	3.6	1.4
2		♀		11.5	5.2	10,400	4.0	31.5	53.5	7.5	3.5	0.0
				12.1±.6	5.1±.1	10,193±592	7.0±1.5	31.7±2	47.7±3.4	8.0±0.9	4±1.8	1.4±1.2
6	32-36 days	♂	Durocs, Chester White, and Duroc Chester White cross	12.1	6.9	15,366	9.7	32.0	50.8	6.1	0.7	0.7
4		♀		13.3	7.0	12,960	13.6	31.0	48.2	5.2	1.0	1.0
				12.7±.47	7.0±.2	14,273±1,293	11.5±1.4	31.5±2	49.6±4	5.7±.8	0.8±.3	0.8±.3
15	3 mo	♂	Durocs, Chester White, and Duroc Chester White cross	11.0	6.7	17,011	9.7	26.5	51.8	2.8	8.1	0.4
15		♀		11.2	6.5	19,421	8.1	24.0	57.7	2.3	6.1	0.6
				11.1±.16	6.6±.2	18,265±829	8.9±.8	25.2±1	54.8±2.6	7.4±4	7.3±.9	0.5±.1
3	5 mo	♂	Hampshires		5.2	12,250	10.3	20.0	61.0	7.7	1.0	0.0
2		♀			5.0	13,077	7.6	22.9	57.7	9.3	2.2	0.6
					5.1±.02	12,871±457	8.3±1	22.2±2.7	58.7±4	9.1±1.5	7±7	6±2

* Data courtesy of W. D. Baten, Experiment Station Statistician, Michigan State University, East Lansing

† Significant difference between sexes

Fig 21
- eosinophilic metamyelocyte
- basophil (2)
- neutrophil showing sex chromatin ("drumstick") (3)

Fig 22
- neutrophil (2)
- lymphocyte (3)
- platelets (1)
- eosinophil (4)
- monocyte (5)

Fig 23
- plasmocyte (1)
- platelets (2)

Fig 24
- eosinophilic band cell (1)
- neutrophil
- progranulocyte (3)
- erythroblast (4)
- erythroblast (5)

Fig 25
- normoblast shedding its nucleus (1)
- degenerating cell (2)
- neutrophilic myelocyte (3)
- lymphocyte (4)
- basophilic metamyelocyte (5)

Fig 26
- neutrophilic metamyelocyte (1)
- eosinophilic myelocyte (2)
- neutrophil band (3)
- same as no 3 (4)
- neutrophilic metamyelocyte (6)
- myeloblast (7)
- lymphocyte (8)
- neutrophilic myelocyte (9)
- segmented neutrophil with false "drumstick" (10)

Fig 27
- eosinophil band cell (1)
- late erythroblast (2)
- degenerating cell (3)
- lymphocyte (4)
- late erythroblast (5)

Fig 28
- myeloblast (1)
- promyelocyte (2)
- erythroblast (3)

Fig 29
- megakaryocyte
- note platelets being pinched off at 2

Fig 21
1 — eosinophilic metamyelocyte
2 — basophil
3 — neutrophil showing sex chromatin (drumstick)

Fig 22
1
2 — neutrophil
3 — lymphocyte
4 — eosinophil
5 — monocyte
platelets
1

Fig 23
1 — plasm
2 — platel

Fig 24
1 — eosinophilic band cell
2 — neutrophil
3 — progranulocyte
4 — erythroblast
5 — erythroblast

Fig 25
1 — normoblast shedding its nucleus
2 — degenerating cell
3 — neutrophilic myelocyte
4 — lymphocyte
5 — basophilic metamyelocyte

Fig 26
1 — neutrophil c metamyelocyte
2 — eosinoph l myelocy
3
4 — same as no 3
5 — neutrophilic metamyelo
6
7 — myelobl
8
9 — lymphocyte
10 — segmented neutrophil with false drumstick
neutrophilic myelocyte

Fig 27
1 — neutrophilic band cell
2 — late erythroblast
3 — degenerating cell
4 — lymphocyte
5 — late erythroblast

Fig 28
1 — myeloblast
2 — proplasmocyte
3 — erythroblast

Fig 29
megakaryocyte
note platelets being pinched off at 2
1
2

f̶ig 2 1 2 2 2 3—adult female blood

served only small granules Venn found the large lymphocyte stained lighter and contained more cytoplasm Sizes given varied from 8 5 to 14μ Kohanawa (1928) determined that the 21 6 per cent of small lymphocytes comprised 90 4 per cent of all the lymphocytes while only 2 3 per cent of the total lymphocytes were of the large variety According to Kohanawa the small lymphocytes varied in size from 5 5 to 11 0 μ and the large lymphocytes from 12 1 to 17 6μ with an average of 14 3μ According to this same investigator 4 per cent of the small and 5 8 per cent of the large lymphocytes exhibited azure granules Vacuolated degenerating forms were extremely rare According to Senftleben (1919) lymphocytes are easily confused with monocytes because of the large size of lymphocytes in pigs (6 8 15 3μ)

In studying lymphocytes and monocytes in our own preparations the similarity between these cells as reported by Senftleben (1919) was confirmed However the nucleus of the lymphocyte stains darker than that of the monocyte and is usually spherical or only slightly indented (Fig 2 2) The cytoplasm tends to stain more blue than the monocyte cytoplasm The large lymphocytes have an increased cytoplasmic nuclear ratio Azurophilic cytoplasmic granules of various sizes are not uncommon

Monocytes The average size of the monocyte reported by Fraser (1938) was 7–8μ by Venn (1944) 13 5μ and Kohanawa (1928) 9 9–15 4μ – 38 9 per cent of which were 13 2μ Venn (1944) described the shape of the nucleus as spherical oval reniform horseshoe shaped and convoluted Fraser found some with as many as three lobes All investigators described azurophilic granules in the cytoplasm

The monocyte may be identified by its large size, its pale staining in comparison with the lymphocyte and the small azurophilic granules uniformly distributed in the grayish blue cytoplasm (Fig 2 2) Small vacuoles also frequently characterize the cytoplasm The most typical nuclear form is the horseshoe shape but all the forms described by Venn (1944) may be observed

Eosinophils The eosinophil nucleus was described as bilobate (Giltner 1907) and 1 to 4 lobed (Palmer 1917a) Venn (1944) stated that the stab form nucleus predominated and that he had never seen a nucleus with more than 2 lobes Fraser (1938) observed young nuclear forms frequently bilobate forms occasionally and rarely 3 lobed nuclei Hirshfeld (1897) found many eosinophils with a spherical nucleus Kohanawa (1928) observed 60 eosinophils and found 8 with 1 41 with 2 9 with 3 2 with 4 and 0 with 5 segments Fraser found large clear cut granules Kohanawa found the granules to be a little larger than those of man and ruminants Senftleben (1919) stated that the eosinophils of swine have particularly fine granules and have a juvenile nucleus According to Rossi (1953) an eosinopenia occurs in castrated animals He related this to a hypertrophy of the cortex of the adrenal gland found in castrates Giltner (1907) counted about 100 granules in each cell According to Venn (1944) the granules do no obscure the nucleus Venn found the average size to be 12μ and Kohanawa (1928) 10 7μ

The eosinophils of swine blood although few in number are readily identified by the eosinophilic granules (Fig 2 1) A comparison of these granules with those from the ruminants proved that they are slightly larger A count of 100 eosinophils revealed 32 per cent with a band nucleus 49 per cent with 2 segments and only 19 per cent with 3 or more segments

Basophils This cell type was considered by Fraser (1938) as the largest of all the cells in the pig He described two types one distended with dark staining granules and the other rarer with fewer and smaller basophilic granules Venn (1944) observed basophilic granules and averaged 12 coarse basophilic granules and averaged 12 μ Kohanawa (1928) gave the average size of 13 2μ Senftleben (1919) described fine basophilic granules

Basophils are rare and difficult to find in a smear but are easily recognized by the basophilic granules which pack the cell and obscure the nucleus (Fig 2 1)

Plasmocyte Fraser (1938) found only one plasma cell in all his slides While Kohanawa (1928) observed this cell in the horse ox sheep and dog he did not find any in swine

This cell type is extremely rare and best located by low power microscopy Its bright robin s egg blue cytoplasm and eccentrically located nucleus distinguish it Higher mag nification reveals a halo about the nucleus and a cartwheel pattern of the nuclear chromatin is usually discernible (Fig 2 3)

Platelets

According to Fraser (1938), the platelets seen in swine blood are small (1–5μ) bodies having a clear plasma limited by a membrane Each contained from 1 to 2 or 40 bluish staining granules The platelets were often clumped together but individual platelets could be observed They were spherical or oval and often had tiny pseudopods Hikmet (1926) counted the platelets in five animals and found they varied from 296 568 to 616 320 with an average of 403 643 per cu mm Wirth (1938) listed the platelet value at 300 000 per cu mm Kohanawa (1928) recorded 263 000 platelets per cu mm and found the size varied from 1 1 to 4 9μ with most of them measuring 2 2μ Cartwright et al (1948) listed mean platelet value in low protein control pigs at 414 000 with a range of 310 000 to 420 000 per cu mm Giltner (1907) found that the platelets varied in size from 1–4μ and numbered 200 000 to 500 000 per cu mm Sopena (1941) found 595 000 platelets per cu mm in hogs Typical platelets are shown in Figures 2 2 2 3 and 2 9

BONE MARROW

Introduction

Relatively little research has been done on the bone marrow of domestic animals and swine in particular Schmidt Nielson and Espeli (1941) chemically analyzed the marrow content of five different bones of

cattle and swine Goodman (1952) made a quantitative study of the distribution of lipids in the right femur bone marrow in the pig and found more lipid by weight in the distal marrow in a 2 month old pig but by 6 months the proximal marrow contained more lipid Kohler (1956) has made an extensive study of the blood and bone marrow of the piglet Varićak (1935) studied the macroscopic appearance of the bone marrow of 413 pigs 9 to 12 months old and 87, 1 to 2 years old In the former the coccygeal vertebrae were completely fat filled the sacral only partly filled with fat and the lumbar entirely red bone mar row Between the ages of one and two years there was fat in the lumbar thoracic and even cervical vertebrae but they still contained foci of red marrow

Noyan (1948–49) included seven swine in a study of the bone marrow of farm animals but did not describe the cells

The sites for bone marrow puncture vary with different investigators Kohler (1956) used the sternum in larger pigs but in small or younger pigs preferred the median or lateral tibial tuberosity Noyan (1948–49) found the wing of the ilium a satisfactory puncture site for swine The animal was snubbed and confined in a standing position and bone marrow was obtained without any apparent physical discomfort except the objection to being confined According to Noyan, bone mar row could not be obtained at the crest of the ilium in swine Wintrobe and his group (Cartwright et al 1950) use a standard 16 gauge needle and aspirate mar row from the sternum

Table 2 5 summarizes the available bone marrow data Preliminary investigations by Diener (1957) indicate that work in pro gress at Michigan State University will re sult in a myelogram very similar to those reviewed here

Red Blood Cell Series

Israels (1941) summarized the matura tion of the erythroblast as follows the cell shrinks to one half its original size the basophilic cytoplasm becomes less and less basophilic polychromatophilic and

TABLE 25
MYELOGRAMS OF SWINE

Author	No of Animals	Stem cell (2-4)	Proerythroblast (0-0.5)	Basophilic Erythroblast (1-3)	Normoblast (15-24)	Progranulocyte (0.5-2.6)	Myelocyte (5-11.2)	Metamyelocyte (5-17.9)	Band Nucleus (5-18)	Segmented Nucleus (5-22)	Monocyte (0.5-1.1)	Lymphocyte (20-30)	Plasmocyte (%)	Reticulum Cell (%)	Myeloid-Erythroid Ratio (2.6)
Hjärre, 1943	8	0.9	0.4	1.7	30.2	1.6	N 6.8 E 0.8	N 36.6 E 0.3		N 12.2 E 0.2	0.5	7.0	0.1	0.1	2.6
Cartwright et al, 1948 and 1950	3	0.4	0.4	2.2	polych 13.9 ortho 18.4	2.7	10.9	28.5	25.2	N 15.7 E 2.4 / 18.1	2.2	4.2	0.2	0.6	1.1
Moretti, 1950	8										0.5	12.4	0.6	0.12	1.2
Gallego, 1951			2.31	4.12	polych 22.07 normobl 9.5	2.2	4.8 1.2 0.06	N 12.0 E 2.8	13.0	N 9.2 E 0.75 B 0.0					
Köhler, 1956				3.3	polych 17.5 oxyphil 12.0	0.3	2.2	9.6	38.1	E 4.6 O 8 B 1.4	0.2	5.2		0.9	3-2.51

finally eosinophilic. The nucleus shrinks and the chromatin condenses, resulting in a featureless dark mass."

All stages in this maturation process appear in a smear, and identification is fairly simple. The more primitive cells contain a reddish-purple nucleus containing nucleoli. As the cell develops, the nucleus becomes smaller, the nucleoli disappear, and the chromatin material condenses and stains darker. At the same time the cytoplasm changes from a blue to a steel-gray, then to the orange-pink color characteristic of the adult red blood cell. In the final stages the pyknotic nucleus is extruded from the cell. Several of these stages are illustrated in Figures 2.4, 2.5, 2.7, and 2.8.

Granulocyte Series

Myeloblast. These cells are few in number, only slightly differentiated from the hemocytoblast and very difficult to identify with certainty (Figs. 2.6 and 2.8).

Progranulocyte. The cytoplasm of this cell lacks specific granulation (neutrophilic, eosinophilic, or basophilic), but may contain azurophilic granules. The chromatin is coarser than that of the myeloblast (Fig. 2.4).

Neutrophilic series. This series is characterized by the presence of neutrophilic granules in the cytoplasm. The nucleus changes from the spherical nucleus of the neutrophilic myelocyte (Figs. 2.5 and 2.6) to the oval or indented shape of the neutrophilic metamyelocyte (Fig. 2.6) to the narrow band of the "band" neutrophil (Figs. 2.6 and 2.7) which constricts and segments to become the adult segmented polymorphonuclear neutrophilic leukocyte (Figs. 2.2 and 2.6).

Eosinophilic series. Ringoen (1921) found great numbers of developing and adult eosinophils in swine. He theorized that the granules were all developed at one time. The cells in this series are readily distinguishable by the bright-red granules. These almost obliterate the blue cytoplasm. The nucleus goes through the same changes

in form as the nucleus of the neutrophil (Figs. 2.4 and 2.6).

Basophilic series. These, too, are very characteristic due to the basic staining granules which pack the cytoplasm and even cover the nucleus to the extent that it is difficult to see (Figs. 2.1 and 2.5). Like the other granulocytes, the nucleus may change from a spherical form to the segmented form or it may remain spherical in the adult form.

Agranulocyte Series

Lymphocytes and monocytes. While the adult forms of these cell series are present in bone marrow smears due to the addition of blood, the developmental stages are in the minority. There is probably little question that there are some foci of development of the nongranulocytic series in the bone marrow, but the lymphoid organs furnish most of these cells.

Megakaryocytes

Kingsley (1935) observed that as the megakaryoblasts of the pig developed, specific granules in the cytoplasm became distinguishable from other stem cells. The granules increased in number as the cell developed and tended to appear in groups in the pseudopods. He found that the nucleus changed from spherical to kidney to horseshoe shape, before developing projections which gave it the complicated appearance of the adult. Megakaryocyte counts were made by Moretti (1950), 0.3 per cent, and Gallego (1951), 0.5 per cent (Fig. 2.9).

BLOOD IN DISEASE

Clinical hematology as an aid in the diagnosis of swine diseases has not been used extensively. This is primarily due to the fact that other symptoms are usually more specific, and that much of the swine diagnostics has been done under field conditions where hematological studies are difficult to undertake. Another difficulty encountered is the variability of normal values as well as those associated with

specific disease conditions. Therefore, unless the changes in the blood picture deviate drastically from the normal, one hesitates to depend on such data alone for the diagnosis of swine diseases.

Virus diseases in general cause a decrease in the total white blood cell count. A leukopenia usually occurs in cases of hog cholera infections. Many times other complicating factors such as pneumonia or parasitism alter the blood picture so that a leukopenia no longer exists.

Bacterial infections generally cause a leukocytosis with an increase in neutrophils and a decrease in lymphocytes. Salmonellosis in the acute form may simulate hog cholera infections except for the accompanying leukocytosis. Acute swine erysipelas produces a severe bacterial septicemia, and therefore a leukocytosis.

Parasitism in swine does not alter the blood picture to a great extent. However, eperythrozoonosis in swine is characterized by causing a severe icterus accompanied by an acute anemia. There is a drop in the red blood cell count to one to two million cells/cu. mm. accompanied by the presence of many immature erythrocytes and reticulocytes. The hemoglobin values decrease to 2-4 gm. The white cell count is usually unchanged but may show a leukocytosis. The bone marrow is hyperplastic.

Anemia is defined as the loss of normal balance between the productive and destructive blood process due to a decrease in volume or a decrease in number of red cells or a reduction of hemoglobin content. A hemolytic decrease in baby pigs caused by maternal iso-immunization has been reported by many workers. The primary symptoms observed are a severe jaundice accompanied by a marked weakness and lassitude. Clinically, there is a drop in the total red blood cell count and a decrease in hemoglobin. Blood smears show enlarged, ringed, and basophilic red cells with reticulocyte counts as high as 60 per cent.

Certain nutritional states of swine cause hematologic changes as observed by Wintrobe and his co-workers at the University of Utah. Pteroylglutamic acid deficiency produces a severe macrocytic anemia accompanied by a leukopenia. Copper and iron deficiencies in swine cause a severe microcytic anemia. In iron-deficient animals the erythrocyte survival is normal and the anemia is due to a decrease in hemoglobin synthesis. However, in copper-deficient swine the anemia is a result of a shortened life span of the erythrocytes. Niacin deficiency causes a moderately severe normocytic anemia with no particular change in the white blood cells.

LYMPH NODES

It is a well-established fact that the lymph nodes of swine have a unique histological structure. The cortical and medullary areas are reversed in position, the denser lymphatic tissue containing the germinal centers occupying a central position, while the more loosely arranged medulla-like component is located peripherally. According to Trautmann and Fiebiger (1957), the afferent lymph vessels pass through the capsule at one or several points and traverse the trabeculae to the interior of the node where they empty into the trabecular sinuses. Converging sinuses at the periphery form the efferent vessels which leave the node at several points on the surface. Recent investigations of Bouwman (1957) suggest that the blood vessels enter with the afferent lymphatics, and also at the point where the efferent lymphatics take origin. As a result there is no true hilus as in the nodes of other species. Microscopic hilus-like indentations are visible where the afferent lymphatics enter. These have been referred to as *pseudohili* by some authors. According to Bouwman, the peripheral medulla-like area is similar histologically to the medulla of ordinary nodes, except that the tissue is not arranged in cords. It is comprised of a basic reticular framework containing small sinuses and clumps of cells uniformly distributed. Collagenous and elastic fibers may also be observed in the medulla. Bouwman described a thin capsule containing collagenous and elastic fibers and smooth muscle cells. The chief trabecular system

of the pig lymph node arises at the points where the afferent lymphatics enter. Bouwman's investigations verified those of earlier workers that many small lymph nodes tend to fuse together to form one large node. After studying lymph nodes prepared by various injection methods, Bouwman supported the view that the afferent lymph vessels penetrate deep into the "cortex," and open into trabecular sinuses which in turn join sinuses which continue to the "medulla." The medullary sinuses converge to form the efferent vessels.

In summary, even though the macroscopic arrangement of the pig lymph nodes is in a sense reversed, the basic histological structures are very similar to those of the lymph nodes of other species (Fig. 2.10).

SPLEEN

The spleen is one of the blood-forming and blood destroying organs and because of

these functions, has considerable importance in the metabolism and defensive mechanisms of the body. The spleen acts as a filter for the blood due to the type of open circulation which allows the blood to come in contact with the fixed and free macrophages of the organ.

Capsule and trabeculae. Among our domestic animals the capsule of the pig spleen is next to the horse in thickness (Trautmann and Fiebiger, 1957). The external surface is covered by a serosa which is intimately attached to the capsule. The capsule is made up of connective tissue rich in smooth muscle, and collagenous and elastic fibers. In the pig the muscle fibers are interwoven (Trautmann and Fiebiger, 1957). At the hilus the capsule continues with the entering vessels and forms the thick trabecular vascular sheaths which ramify with the vessels.
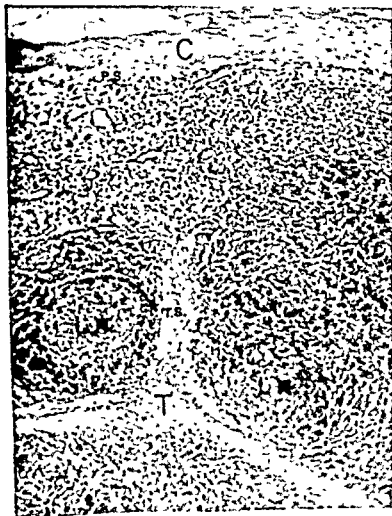
FIG. 2.10—Pig lymph node. Hematoxylin-eosin. X 95.
P.S —peripheral sinus
T.S —trabecular sinus
L.N —lymph nodule
C—capsule
T—trabecula

Heavy trabeculae make up the remainder of the framework of the spleen. They radiate from the capsule and continue to branch and rebranch forming a netlike arrangement which serves as a scaffold for the splenic pulp. According to Tischendorf (1952), muscle is present in the pulp of the pig spleen as single isolated fibers. He reported that these myofibrils are united to each other and to the reticulum cells by special foot plates. This ramified arrangement forms a tridimensional network allowing the myofibrils to continue through several longitudinally connected cells. This pulp musculature has a function antagonistic to the capsule-trabecular system.

The reticular network extends throughout the red and white pulp and marks the border between them as illustrated in Figure 2.11. Trautmann and Fiebiger (1957) reported that the reticulum of the red pulp is especially strong in the pig.

Blood vessels. The main arterial supply of the pig spleen is similar to that of other animals. The splenic artery enters at the hilus, branches with the trabeculae, and continues into the pulp through the splenic corpuscles as the central artery, and then into the red pulp as fine penicilli. The penicilli ramify into the ellipsoids and the arterial capillary segments enter the red pulp and terminate in rounded ampullae. In the pig the red pulp contains primordial veins (Snook, 1950) which anastomose with the trabecular veins and extend to the hilus.

Red pulp. The pig spleen contains considerable white pulp and relatively little red pulp. According to Snook (1950) the red pulp is of the non sinusoidal type having small primordial veins which lead directly from the pulp meshes into collecting veins Trautmann and Fiebiger (1957) reported that the sinusoids of the pig are subordinate to the rest of the red pulp.

Penicilli branch from the central artery and enter the red pulp. At this point they are invested by a large ellipsoid (Schweiger-Seidel sheath) composed of closely

FIG. 2.11—Pig spleen. Two splenic corpuscles surrounded by reticular network. Reticular impregnation. X 100

packed, pale-staining cells embedded in a net of reticular fibers. The ellipsoids of the pig spleen are very well developed and distinct (Fig. 2.12). Snook (1950) reported them to be the second largest (195–62μ) of any of the animals he had studied, exceeded only by that of the mole. Ellipsoids have been reported as nervous structures and muscular organs, but it is generally believed that these structures are a condensation of reticular fibers and reticuloendothelial cells investing a capillary. The exact function of these ellipsoids is unknown, but some believe that they serve as the first filters for the arterial blood since there are openings between the reticulo endothelial cells.

The parenchyma of the red pulp is composed of modified lymphatic tissue in a framework of reticular fibers. In the meshes of the network are free macrophages, all elements of the circulating blood, with the agranulocytes being the most numerous. The lymphocytes which are present in the red pulp originate in the white pulp and migrate by ameboid movement. The macrophages are round or irregularly shaped cells with a vesicular nucleus. Many times these cells engulf erythrocytes in various stages of degeneration. Frequently yellow and brown granules are seen in these cells as a result of red cell destruction. Other cells found in the red pulp include myelocytes, plasmo-
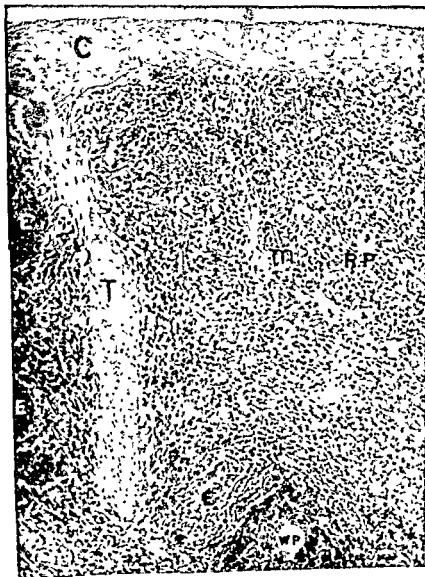
FIG. 2.12—Pig spleen.
Hematoxylin-eosin. X 95.
C—capsule
T—trabecula
E—ellipsoid
M—smooth muscle
R.P.—red pulp
W.P.—white pulp

cytes, erythroblasts, and megakaryocytes Seamer (1956) reported finding particularly prominent megakaryocytes in the red pulp of the spleens of young pigs

White pulp. The white pulp which is associated with the central artery mentioned above is made up of a reticular framework investing free lymphocytes of various sizes These cells are distributed so as to form nodular lymphatic tissue The center of the nodule is pale staining since it contains the young, undifferentiated lymphoblasts while the periphery is dark due to the more mature lymphocytes

REFERENCES

ALBRITTON, E C 1952 Standard Values in Blood W B Saunders, Philadelphia

BARBER, R S, BRAUDE, R, AND MITCHELL, K G 1955 Studies on anaemia in pigs 2 Comparison of haemoglobin levels in blood of pigs reared indoors and outdoors on pasture Vet Rec 67 543

BODDIE, G F 1956 Diagnostic Methods in Veterinary Medicine J B Lippincott Company, Philadelphia

BOUWMAN, F L 1957 Unpublished data Detroit Institute of Technology, Detroit

BRUNER, D W, BROWN, R G, HULL, F E, AND KINKAID, A S 1949 Blood factors and baby pig anemia Jour Amer Vet Med Assn 115 94

BUNCE, S A 1954 Observations on the blood sedimentation rate and the packed cell volume of some domestic farm animals Brit Vet Jour 110 322

BUSH, J A, BERLIN, N I, JENSEN, W N, BRILL, A B, CARTWRIGHT, G E AND WINTROBE, M M 1955a Erythrocyte life span in growing swine as determined by glycine 2-C14 Jour Exper Med 101 451

——— JENSEN, W N, CARTWRIGHT, G E, AND WINTROBE, M M 1955b Blood volume studies in normal and anemic swine Amer Jour Physiol 181 9

——— ATHENS, J W, ASHENBRUCKER, H, CARTWRIGHT, G E, AND WINTROBE, M M 1956 Studies on copper metabolism XIX. The kinetics of iron metabolism and erythrocyte life span in copper deficient swine Jour Exper Med 103 701

CARLE, B N, AND DEWHIRST, W H., JR. 1942 A method for bleeding swine Jour Amer Vet Med Assn 101 495

CARTWRIGHT, G E, FAY, J, TATTING, B, AND WINTROBE, M M 1948 Pteroylglutamic acid deficiency in swine effects of treatment with pteroylglutamic acid, liver extract and protein Jour Lab and Clin Med 33 397

——— PALMER, J., HITCHINGS, G, ELION, G, GUNN, F, AND WINTROBE, M M 1950 Studies of the effect of 2 6 Diaminopurine on the blood and bone marrow of swine Jour Lab and Clin Med 35 518

CRAFT, W A, AND MOE, L H 1932 Statistical observations on weight, hemoglobin and proportion of white blood cells in pigs Jour Amer Vet Med Assn 81 405

DAVIDSON, W M, AND SMITH, D R 1954 Sex differences in the polymorphonuclear neutrophil leucocytes Brit Med Jour 2 6

DIENER, A M 1957 Unpublished data Anatomy Dept, Mich State Univ East Lansing Mich

DIGGS, L W, STURM, D, AND BELL, A 1956 The Morphology of Human Blood Cells W B Saunders Company Philadelphia

DINWIDDIE, R R 1914 Studies on the hematology of normal and cholera infected hogs Ark Exp Sta Bull 120

DOLL, E R, AND BROWN, R. G 1954 Isohemolytic disease of newborn pigs Cornell Vet 44 86

DOYLE, L. P, MATTHEWS, F P AND WHITING R A 1928 Anemia in young pigs Jour Amer Vet Med Assn 72 491

DUKES, H H 1955 The Physiology of Domestic Animals Comstock Publishing Associates Ithaca New York

FRASER A C 1938 A study of the blood of pigs Vet Jour 94 3

GALLEGO, E G 1951 El mielograma normal en la especie porcina An Fac Vet 3 129

GARDINER, M R, SIPPEL, W L, AND McCORMICK, W C 1953 The blood picture in newborn pigs Amer Jour Vet Res 14 68

GARDNER M V 1947 The blood picture of normal laboratory animals A review of the literature 1936 1946 Notes Biochem Res Found, J Franklin Inst., p 26

GILTNER W 1907 The histology and physiology of normal pig's blood Jour Comp Path and Therap 20 18

GOODMAN, D C 1952 Quantitative studies on the distribution of lipids in the bone marrow of the rat, pig and cat Trans Kansas Acad Sci 55 214

GOODWIN, R F W, AND COOMBS, R R. A 1956 The blood groups of the pig IV The A antigen antibody system and haemolytic disease in newborn piglets Jour Comp Path and Therap 66 317

GOODWIN, R F W, AND SAISON, R 1956 The blood groups of the pig III A breed difference in iso antibody response after vaccination with crystal violet swine fever vaccine Jour Comp Path and Therap 66 163

GRADWOHL, R B H 1956 Clinical Laboratory Methods and Diagnosis Vol I C V Mosby Company, St Louis

HANSARD, S L 1956 Residual organ blood volume of cattle, sheep and swine Proc Soc Exper Biol and Med 91 31

———, BUTLER, W O, COMAR, C L, AND HOBBS, C S 1953 Blood volume of farm animals Jour Anim Sci 12 402

——— SAUERLICH, H E, AND COMAR, C L 1951 Blood volume of swine Proc Soc Exper Biol and Med 78 544

HEWITT, E A 1932 Certain chemical and morphologic phases of the blood of normal and cholera infected swine Iowa State Coll Jour Sci 6 143

HIKMET, P 1926 Die Blutplättchen biem gesunden und kranken Pferd, Hund und Schwein Arch wiss prakt Tierheilk 55 222

HIRSCHFELD, H 1897 Vergleichende Morphologie der Leukozyten Virch Arch 149 22

HJARRE, A 1943 Om sternalpunktion och den normala benmargsbilden hos huskjuren Skand vet tidskr 33 457

HOLMAN, H H 1956 Clinical hematology In G F Boddie, Diagnostic Methods in Veterinary Medicine J B Lippincott Company, Philadelphia p 322

HUDSON, A E A 1955 Fragility of erythrocytes in blood from swine of two age groups Amer Jour Vet Res 16 120

ISRAELS, M C G 1941 The hemoglobinization of erythroblasts Jour Path and Bact 52 361

JENSEN, W N, BUSH, J A ASHENBRUCKER, H, CARTWRIGHT, G E, AND WINTROBE, M M 1956 The kinetics of iron metabolism in normal growing swine Jour Exper Med 103 145

JONES, J M, SHIPP, M E, AND GONDER, T A, JR 1936 Changes occurring in the blood picture during fetal life Proc Soc Exper Biol and Med 34 873

KENNEDY, W P AND CLIMENKO, D R 1931 Studies on the Arneth count 18 The normal count in various mammals Quart Jour Exper Physiol 21 253

KERNKAMP, H C H 1932 The blood picture of pigs kept under conditions favorable to the production and to the prevention of so called "anemia of suckling pigs" Univ Minn Agr Exp Sta Tech Bull 86

——— 1933 Results in the use of fresh and oxalated blood of swine when making cellular counts and hemoglobin determinations Jour Amer Vet Med Assn 36 666

KING, W E, AND WILSON, R H 1910 Studies on hog cholera and preventive treatment Kan Agr Exp Sta Bull 171, p 139

KINGSLEY, D M 1935 The development of the megakaryocyte in the pig Anat Rec 61 (suppl) 29

KOHANAWA C 1928 Beiträge zur vergleichenden Morphologie des Blutes der gesunden Haus saugetiere Fol Haem 36 174

KOHLER H 1956 Knochenmark und Blutbild des Ferkels 1 Das gesunde Ferkel 2 Das Ferkel mit spontaner Anamie Zentralbl f Veterinär Med 3 359 460

LEWIS, L L, AND SHOPE, R E 1929 The study of the cells of the blood as an aid to the diagnosis of hog cholera Jour Amer Vet Med Assn 74 (n.s 27) 145

LUKE D 1953a The reaction of the white blood cells at parturition in the sow Brit Vet Jour 109 241

——— 1953b The differential count in the normal pig Jour Comp Path and Therap 63 346

——— 1953c The effect of adrenocorticotrophic hormone and adrenal cortical extract on the differential white blood cell count in the pig Brit Vet Jour 109 434

MEYER, S 1924 Die Blutmorphologie einiger Haus und Laboratoriumstiere unter physiologischen und pathologischen Bedingungen Fol Haem 30 195

MOLINA R R, AND GONZALEZ J O 1940 Puerto Rico Jour Pub Health Trop Med 15 383 (Reported by Gardner, 1947)

MORETTI B 1950 Attignamenti funzionali del midollo sternale nella pesta suini pura speri mentale Arch Vet Ital 1 139

MUHRER, M E, HOGAN, A G, AND BOGART, R. 1942 A defect in the coagulation mechanism of swine blood Amer Jour Physiol 136 355

NOYAN, A 1948-49 A study of bone marrow in farm animals Abst Doct Diss Ohio Univ No 60 p 237

OGLESBY, W T, HEWITT, E A, AND BERGMAN, H 1931-32 Certain chemical and morphologic phases of the blood of normal and cholera infected swine II Certain morphological phases Iowa State Coll Jour Sci 6 227

ORFEI Z 1950 Studio del quadro ematico periferico nella pesta pura dei suini Arch Vet Ital 1 181

OSGOOD, E E 1940 A Textbook of Laboratory Diagnosis The Blakiston Company Philadelphia

——— AND ASHWORTH C 1937 Atlas of Hematology J W Stacey Inc. San Francisco

PALMER, C C 1917a Morphology of normal pigs blood Jour Agr Res 9 131

——— 1917b Effects of muscular exercise and heat on the blood and body temperatures of normal pigs Jour Agr Res 9 167

PAYNE L. C. 1952 Useful Physiological Data (a compilation) Fort Dodge Laboratories Inc Fort Dodge Iowa

REGNER A 1923 Ein Beitrag zum Blutbilde des gesunden und kranken Schweines und dessen Verwertung bei Diagnose von Rotlauf Schweinepest und Schweineseuche Wien tierarztl Monatschr 10 97

RINGOEN A L 1921 The origin of the eosinophil leucocytes of mammals Fol Haem 27 10

ROSSI, G 1953 Sul tisso ematico di granulociti eosinofili in suini sani razza Ann Fac Med Vet Pisa 6 110

SCARBOROUGH R A 1931 32 The blood picture of normal laboratory animals Yale Jour Biol 3 63 168 267, 359 431 547 4 69 119 323

SCHMIDT NIELSON, S AND ESPELI A 1941 Bone marrow of cattle and swine Kgl Norske Videnskab Selskabs Forh 14 13 Abst Chem Zentralbl 1 1823 1942

SEAMER J 1956 Piglet anemia a review of the literature Vet Rev and Annot 2 79

SENFTLEBEN O 1919 Das Blutbild des gesunden Schweines Monatsh f prakt Tierheilk 30 289

SMITH E. M AND CALHOUN M L 1956 Observations on the occurrence of sex chromatin (drumsticks) in the polymorphonuclear neutrophil leucocyte of domestic and laboratory animals Paper presented 58th Meet Mich Acad Sci Arts and Letters Ann Arbor

SMITH F 1912 A Manual of Veterinary Physiology Alexander Eger Chicago

SNOOK T 1950 A comparative study of the vascular arrangements in mammalian spleens Amer Jour Anat 87 31

SOPENA I 1941 Determinación del número normal de plaquetas sanguineas en algunas especies domesticas Univ Buenos Aires Rev Fac. Agron y Vet 9 73 (Biol Abst 18 565 No 5069 1944)

SPLITTER E J 1953 Observations on an erythrocytic inclus on in swine Amer Jour Vet Res 14 575

STAUB H 1924 Blood samples from small piglets Berl Munch tierarztl Wschr 67 188

SWENSON M J GOETSCH D D AND UNDERBJERG G K L 1955 The effect of the sow s ration on the hematology of the newborn pig Proc. Amer Vet Med Assn p 159

——, UNDERBJERG G K L. GOETSCH D D AND AUBEL C E 1957 Hematology histo pathology and growth of newborn p gs implanted subcutaneously with Bacitracin pellets Unpublished data Dept of Physiol Kansas State Coll Manhattan

SZENT IVANYI TH, AND SZABO ST 1954 Blood groups in pigs Acta Vet 4 429

TISCHENDORF F 1952 D e Pulpamuskulatur der Milz und ihre Bedeutung Zeitschr Zellforsch mikr Anat 36 2 (Biol Abst 29 1340 No 13518 1955)

TRAUTMANN A AND FIEBIGER J 1957 Fundamentals of the H stology of Domest c Animals Translated and revised from 1949 German edit on by Robert E Habel and Ernst L Biber stein Comstock Publishing Associates Ithaca New York

UNDRITZ E 1952 Sandoz Atlas of Haematology Sandoz Pharmaceutical Ltd Basle Sw tzer land

VARIĆAK D 1935 Zur Kenntnis des Markes der Rumpfknochen Untersuchungen zwecks klin ischer Auswertung an Pferd Rind Schwein Hund und Katze Arch wiss prakt Tierheilk 78 461

VENN J A J 1944 Variations in the leucocyte count of the pig during the first twelve weeks of life Jour Comp Path and Therap 54 172

WINTROBE, M M 1951 Clinical Hematology 3rd ed Lea & Febiger Ph ladelphia

—— AND SHUMACKER H B 1936 Erythrocyte studies in the mammalian fetus and newborn Erythrocyte counts hemoglobin and volume of packed red corpuscles mean corpuscular volume d ameter and hemoglobin content and proportion of immature red rels in the blood of fetuses and newborn of the pig rabbit rat cat dog and man Amer Jour Anat 58 313

WIRTH D 1938 Die besondere Reaktionsweise der hamatopoet schen Organssteme bei unseren Haussaugetierarten Dreizehnter int tierarztl Kongress Zurich Interlaken 1 273

—— 1950 Grundlagen einer klinischen Haematolog e der Haustiere Urban und Schwartzen berg Wien Innsbruck

——. ROSMANN F AND BENNDORF G 1939 Stud en zur artspezifischen Reaktion der hamatopoetischen Organsysteme (V Schwein) Fol Haem 61 1

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CHAPTER 3

Physiology

Little is known of the physiology of swine Much of the available information is derived from studies designed to answer problems related to animal husbandry and not to obtain fundamental physiological data For example, because of its applied importance in the rearing of swine there is much more information on environmental physiology than on basic aspects of respiratory or cardiovascular physiology In some instances only isolated facts have been accumulated on particular organs and systems, and therefore it is impossible to develop any correlated discussion at this time For these reasons, the chapter presented here is not a balanced one and the amount of space devoted to the various subjects does not necessarily reflect their physiological importance but rather the relative availability of information The material is confined almost entirely to specific data on swine, and no attempt has been made to discuss general physiological problems as they might be related to this animal

CARDIOVASCULAR SYSTEM

Dukes (1955) states that the heart rate of the resting pig varies from 60 to 80 beats per minute This is a much lower value than that obtained by Platner *et al* (1948), who studied heart rate by means of an electrocardiograph and reported a mean rate of 182 per minute However, this higher rate may be in part due to the

animals' struggling Luisada *et al* (1944), in a study on a limited number of pigs of various weights (45 to 400 pounds), recorded heart rates of 120 to 135 per minute

Dukes and Schwarte (1931a) found the mean pressure in the carotid artery of a group of 14 pigs under local anesthesia to be 169 mm Hg In the same report these workers described the influence of certain nervous and nonnervous factors on the cardiovascular apparatus of the pig Stimulation of the central end of the right vagus nerve usually caused a rise in blood pressure while stimulation of the central end of the left vagus had little or no effect Stimulation of the peripheral end of the splanchnic nerve led to a rise in blood pressure, sometimes as much as 100 per cent In the pig the cervical sympathetic nerve on both sides is separate from the vagus nerve

Platner *et al* (1948) studied the electrocardiogram of pigs which were between 60 and 90 days old and reported the following average values (in seconds) for duration of the various waves P wave, 0 047, P-R interval, 0 074, QRS wave, 0 044, Q-T interval, 0 331 These figures are not in general agreement with those of Luisada *et al* (1914), who obtained the following values (in seconds) based on a limited number of pigs P wave, 0 08, P-R interval, 0 12 to 0 14, QRS wave, 0 06, Q-T interval, 0 22 to 0 28 Luisada's group

(1944) also recorded heart sounds in pigs by means of phonocardiograms According to their data the first sound lasts from 0 12 to 0 14 seconds and is composed of three or four quick and large vibrations followed by some others which are smaller and slower The second sound occurs shortly after the end of the T wave and lasts 0 04 to 0 06 seconds

Sporri (1954a) found that the relative duration of diastole to systole was shorter in the pig than in any of the other animals studied (horse dog elephant, cattle sheep) In a later report Sporri (1954b) proposed that the predisposition of pigs to death from acute heart failure may be related to the very short period of diastole He reasoned that because of compression of the myocardial vessels during systole the flow of blood to the heart muscle is confined almost entirely to the period of diastole A short diastolic period is thus unfavorable for exchange of metabolites between blood and musculature and the recovery processes in the muscle fiber during diastole are endangered With faster heart rates the diastolic period is reduced proportionately more than the systolic period and for this reason a tachycardia produces more unfavorable conditions for metabolic exchange and recovery Tachycardia is thus a particular danger for the pig

Thiamine deficiency in the pig results in various changes in the electrocardiogram and cardiac activity (Wintrobe et al 1943) These include bradycardia prolonged P-R interval second degree atrioventricular block abnormalities in P waves nodal and ventricular premature beats auriculoventricular dissociation complete heart block with an ectopic ventricular rhythm and auricular fibrillation The changes observed in the thiamine deficient pig appear more pronounced than those seen in other animals or man

RESPIRATORY SYSTEM

The breathing rate in pigs varies from 8 to 18 per minute Dukes and Schwarte (1931b) made a limited number of observations on respiratory reflexes of pigs in which results were

quite inconsistent Stimulation of the central end of the vagus nerve usually caused complete inhibition of breathing Crushing of the vagus or stimulation of the central end of the sciatic nerve gave variable results while occlusion of the carotid arteries had little or no effect on breathing

Young pigs tend to huddle together for warmth and if observed closely can some times be seen to shiver with each inspiration and then to lie quietly without shivering at intervals Cort and McCance (1953) studied the mechanism of shivering and its relationship to breathing in a series of acute experiments If cold air was introduced directly into the trachea shivering movements occurred during inspiration followed by a pause during expiratory rest If the stimulus was continued for some time, the shivering increased in amplitude but lost its respiratory rhythm Cutting the vagus bilaterally abolished the rhythmic response These experiments seem to indicate the existence of a temperature receptor in the walls of the trachea bronchi or bronchioles and demonstrate that vagal reflexes are important in the initial rhythmic response associated with inspiration

GASTROINTESTINAL TRACT

Under natural conditions food is carried to the mouth of the pig largely by action of the pointed lower lip As an aid in obtaining food the animal may dig up the ground with its snout When pigs are prevented from rooting prehension of food is accomplished by the teeth tongue, and characteristic movements of the head

The esophageal region of the pig's stomach is limited to a small area around the cardia The cardiac gland zone is quite extensive occupying the entire left extremity of the stomach The fundic zone is large and the pyloric gland zone extends to the pylorus and is similar to the pyloric region in the dog and man

Saliva of the pig contains an amylase but its amylolytic activity is much less powerful than that of human saliva Amylase is not found in gastric secretions but pepsin and lipase are present The normal gastric emptying time in the pig is pro-

longed and it requires a fast of many hours to empty the stomach completely As a result digestion and absorption are almost continuous processes (Link 1953a)

Neimeier (1941) studied gastrointestinal motility in pigs with the aid of a radio opaque contrast medium and roentgen rays The duodenum is very active for the first hour after feeding after which the region becomes less active for about 30 minutes and then becomes quite active once more Ingesta quickly reach the jejunum which rapidly fills but the passage of food through the jejunum is much slower than through the duodenum Approximately 1¼ to 2 hours after feeding the jejunum shows definite motor activity and within 6 to 10 hours it is empty The jejunum shows a varied type of motility including pendular movements rhythmic segmentations to and fro movements of the entire jejunal contents and large and small peristaltic waves The ileum exhibits an irregular motility At times the ileocecal sphincter closes and stops the ingesta but at other times the ingesta pass freely into the colon In most instances the material enters the cecum but occasionally the colon receives the material before it enters the cecum Cecal motility diminishes 6 to 8 hours after feeding approximately 14 to 16 hours after feeding balls of feces begin to leave the colon

The type of motility of the colon can be associated with its peculiar anatomical arrangement (Trautmann and Asher 1940) The spiral portion shows pendular bending and tonic contractions in its individual segments while the terminal portion shows only peristaltic waves Antiperistaltic waves appear to be absent from the colon

RENAL SYSTEM

Ellenberger and Scheunert (1925) state that the average daily volume of urine excreted by adult pigs is 4 liters with a range of 2 to 6 liters In more recent studies on boars Green (1914) found that the amount of urine excreted over a 48 hour period may show wide variations In a total of 486 samples collected from 18 boars there were 58 instances in which no urine was

collected during a 48 hour interval On the other hand up to 15 600 ml of urine were collected in some cases The average excretion for 48 hours was 4 287 ml Significant variations were found in different lines of pigs

Grace et al (1951) experimentally produced uremia in pigs by ligating both ureters This resulted in an increased blood concentration of nonprotein nitrogen urea nitrogen uric acid creatinine and allantoin The increase in uric acid was more pronounced in younger pigs than older indicating that the former may not as yet have become adapted to convert uric acid to allantoin

The average specific gravity of urine from swine is 1 012 with a range of 1 010 to 1 050 (Ellenberger and Scheunert 1925)

MILK AND MILK PRODUCTION

Composition of Milk

Chemical composition Hughes and Hart (1935) reviewed the literature on the composition of sows milk and calculated a mean value of 18 2 per cent for total solids In studies of their own on two sows they obtained values for colostrum at parturition of 31 9 per cent total solids 16 64 per cent protein and 0 61 per cent ash For normal milk the values were 17 76 per cent total solids 6 34 per cent protein and 0 96 per cent ash In general these values are in good agreement with those reported more recently by Heidebrecht et al (1951) on a larger series of animals except that these workers found an average of approximately 20 per cent for total solids Sows milk contains an average of 9 58 per cent fat and 4 62 per cent lactose although the fat content may show considerable daily variation (Perrin 1954) Unlike the situation in most domestic animals the per cent of ash in sows colostrum is lower than that in normal milk However, as lactation progresses there is a gradual rise in the content of ash (Perrin 1955)

Any critical studies on the composition of milk must take into account the effect of the plane of nutrition and the stage of

lactation This was indicated by the work of Bowland et al (1949b), who studied and compared the composition of milk from sows on pasture with those on dry lot feeding The results of this study are given in Table 3 1 These workers found that on the third day of lactation there is a sudden rise in the fat content of milk This apparently is the result of the sow's converting body fat into milk at a very rapid rate after farrowing The protein content of milk tends to decline up to the third week after farrowing at which time it begins a gradual rise The values obtained by Bowland et al (1949b) are in good agreement with those reported earlier by Braude et al (1947) and in a more recent study by Barber et al (1955)

Vitamin content. The vitamin A level of sow's colostrum and early milk follows a different pattern than that of the cow (Bowland et al, 1949a) In the cow the vitamin A content shows a rapid fall during the first three milkings and reaches a normal range by the fourth or fifth day In the sow the vitamin A level is still nearly double that of normal milk at the end of the first week of lactation In sows on pasture the vitamin A level for first and third day colostrum averages 143 6 μg per 100 ml Colostrum from sows on pasture compared to those on dry lot shows little difference in vitamin A content, but the vitamin level in normal milk of pastured animals is nearly 50 per cent higher than those on dry feed Those on pasture average 52 7 μg of vitamin A per 100 ml of milk while those on dry feed show an

average of 34 8 μg per 100 ml (Bowland et al, 1949a)

The Vitamin C level of sow's milk is considerably higher than that of many other animals Bowland et al (1949a) found levels of 18 8 mg per 100 ml in colostrum and 10 4 mg per 100 ml for normal milk of sows on pasture Sows on dry lot feeding showed values of 24 6 mg per 100 ml and 12 2 mg per 100 ml respectively Holmes et al (1943) reported average values of 2 18–2 69 mg of vitamin C per 100 ml of cow's milk

Davis et al (1951) obtained the following average values (given in μg per ml) for certain members of the vitamin B group in sow's colostrum thiamine 0 6 riboflavin 5 0, niacin 1 7, and pantothenic acid 0 7 The thiamine content of normal milk does not differ appreciably from that in colostrum, but the riboflavin content is lower and the niacin and pantothenic acid levels are higher

Milk Production

Milk yield. In studies on a group of Large White pigs Barber et al (1955) found that the average daily yield of milk was 13 71 lbs (range 11 6–15 7 lb) In this study the milk yield was computed by weighing pigs before and after suckling The pigs were allowed to nurse at hourly intervals, since under natural conditions this is the average interval between nursings When suckling is permitted at hourly intervals, the daily milk yield is considerably greater than when it is allowed only every two to three hours

TABLE 3 1

AVERAGE COMPOSITION OF MILK FROM SOWS ON PASTURE AND DRY LOT FEEDING *

	Pasture Sows		Dry Lot Sows	
	First Day Colostrum	Later Milk	First Day Colostrum	Later Milk
Total solids per cent	22 81	19 47	22 81	20 69
Solids not fat per cent	15 92	13 16	17 21	13 38
Protein, per cent	11 25	7 09	14 29	7 42
Lactose, per cent	2 89	5 18	3 42	5 08
Ash, per cent	0 72	0 99	0 73	0 98

* Taken from data of Bowland et al (1949b)

The peak yield of milk is reached between the second and fourth weeks of lactation (Barber *et al*, 1955) During the first four weeks of lactation pigs probably obtain milk every time they suckle when this is permitted at hourly intervals In later stages of lactation the pigs may not obtain milk at each nursing

Milk ejection Sows will not voluntarily eject milk in response to any stimulus other than that provided by the suckling of pigs Supposedly the nursing process sets up a neural stimulus which initiates the release of oxytocin by the posterior pituitary gland The hormone is carried in the blood stream to the mammary gland where it produces a let down of milk by causing contraction of the myoepithelial cells around the alveoli It is possible to hand milk or machine milk sows if oxytocin is first injected (Braude and Mitchell 1952) The amount of milk obtained after injection of oxytocin depends upon the dose of the hormone employed (Braude and Mitchell 1950) A dose of 10 International Units (I U) results in the ejection of approximately 104 ml of milk from a single mammary gland while 1 I U produces a flow of 41 ml which is approximately equivalent to the amount of milk obtained from a single gland by a pig at a natural nursing

In addition to the effect of oxytocin other hormones or chemicals may have a marked effect on milk flow in the sow The injection of acetylcholine results in a let down of milk It is probable that this is a result of the direct action of acetylcholine on the mammary gland although the possibility of its acting through the pituitary gland has not been completely eliminated (Whittlestone and Turner 1952) Epinephrine has an inhibitory effect on milk flow (Braude and Mitchell 1952 Whittlestone 1954) The injection of 0 2 mg of epinephrine immediately before administration of 0 5 I U of oxytocin will suppress the rise in intramammary pressure and the subsequent milk flow produced by the latter hormone (Whittlestone 1954)

Suckling Behavior of Pigs

Barber *et al* (1955) studied the suckling behavior of pigs and found that under natural conditions the time between successive nursing periods was 60 to 75 minutes The first phase of each suckling period is characterized by the pig's vigorous nosing of the udder as soon as the sow assumes a nursing position The length of this phase increases as pigs get older and this may mean that the secretion of the let down hormone from the posterior pituitary is delayed as lactation advances During the second phase of nursing the pigs suddenly become quiet and at this time they obtain milk The average time during which milk ejection occurs was found to be 18 5 seconds During the first part of lactation pigs may show considerable fighting for individual teats but gradually they assume a set position so that usually by the end of the first week of lactation the two anterior teats and the two posterior teats are suckled by the same pigs Later on (usually by two weeks) the middle teats are occupied at each nursing by the same pigs Pigs seem to show a preference for the anterior teats and in general these teats yield the most milk

REPRODUCTIVE SYSTEM

Reproductive System in the Female

Estrous cycle The sow is a polyestrus animal with breeding cycles continuous over the entire year The length and character of the estrous cycle has not been studied as extensively in sows as in many other domestic animals and critical information on the length of various parts of the cycle is lacking The mean length of the cycle is 21 days with a standard deviation of 2 5 days Barker (1951) citing earlier studies on the character of the corpus luteum divided the various stages of the cycle as follows Metestrus follows ovulation and lasts for a period of three days diestrus occupies the period from the fourth until the 17th day after ovulation and proestrus lasts from the 18th day until *ovulation Estrus occupies a period of one*

to five days with an average of two or three days, and ovulation occurs between 36 and 48 hours after the onset of estrus (Pomeroy, 1955).

Various studies have been conducted to determine the age at which puberty (i.e. the first heat period) occurs in gilts. Phillips and Zeller (1943) reported than in small-type pigs (average weight 189.5 pounds) the first estrus occurs at an average of 207.8 days, while larger-type pigs (average weight 199.3 pounds) exhibit their first heat at an average of 198.7 days. Robertson et al. (1951) found no significant difference in the age at which Poland China gilts and Chester White gilts reached puberty. The average figures for the two breeds were 201 days and 204 days, respectively. It was observed that Poland China gilts were significantly heavier (average weight 224 pounds) than Chester White gilts (average weight 212 pounds) at puberty. The season at which pigs are born may influence the age at which they reach puberty. Gilts born in the spring (i.e. the natural farrowing season) are slower to reach sexual maturity than those born at other seasons (Wiggins et al., 1950; Robertson et al., 1951).

There is considerable disagreement concerning the incidence of postpartum estrus in pigs. Smith (1937) and Hart (1949) state that sows may show estrus a few days, usually the third, after farrowing. On the other hand, Asdell (1946) indicates that sows do not usually come into heat until after the end of lactation. Warnick et al. (1950) studied a group of 36 sows starting immediately after farrowing and found that 18 of them showed a heat period between one and five days after parturition. Only two of the animals ovulated, and since these were the only sows in the group which did not nurse their pigs, it appeared that suckling might inhibit ovulation. However, subsequent observations by Warnick et al. (1950) and by Baker et al. (1953) failed to indicate any such effect. The latter group of workers studied 29 sows and reported that 17 of them showed heat between the first and third days after farrowing. None of the sows conceived and it was concluded that ovulation does not ordinarily occur at the postpartum heat. Warnick et al. (1950) believe that an extra-ovarian source of estrogen is probably responsible for the postpartum heat since the ovaries of sows at this time do not contain follicles large enough to be a source of much estrogen. Estrus can be rather consistently induced in sows by the injection of 700 to 1,500 International Units of equine (serum) gonadotropin between the 39th and 65th days of lactation (Cole and Hughes, 1946).

Ovarian changes. Immediately a f t e r ovulation only small ovarian follicles are found. These gradually enlarge and by the time of proestrus reach a size of approximately 4–6 mm. in diameter. During proestrus the follicles grow to a size of 8–12 mm. and by the time of estrus there are 15 to 40 large follicles present, most of which rupture at the time of ovulation. Casida (1935) determined that follicles first appear on the ovary when gilts have reached an age of approximately 80 pounds and are 15 to 16 weeks old. This would place the appearance of follicles about midway between birth and the onset of the first heat period. The estrogen content of large Graafian follicles is approximately 900 Rat Units (R.U.) per kilogram of liquor folliculi (Allen and Doisy, 1927). On the basis of cytochemical studies Barker (1951) concluded that the cells of the theca interna of follicles are the site of production of ovarian steroid hormones, presumably estrogen.

Hemorrhage rarely occurs into the cavity of a ruptured Graafian follicle. The corpora lutea are pinkish when fully developed but become yellowish as degeneration occurs. A detailed description of the formation and development of the corpus luteum in sows is found in the report of Corner (1919). Studies by . Kimura and Cornwell (1938) have shown that during the first three days following ovulation the corpora lutea contain large amounts

of progesterone The level of the hormone rises until 15 days after ovulation at which time retrogression of the corpora lutea occurs and the progesterone content falls rapidly If pregnancy occurs, the hormone content continues to rise sharply until the 20th day, and the level remains high or may rise further until approximately the 105th day of pregnancy, after which it falls rapidly The phospholipid content of the corpus luteum varies directly with the activity of the gland (Bloor *et al*, 1930) From the fifth to the tenth day of the estrual cycle there is a marked increase in phospholipid content During the period of active function of the corpus luteum (tenth to fourteenth day) the level remains high but does not show a further increase and from the fourteenth to seventeenth day there is a rapid decrease in phospholipid content

Uterine and vaginal changes The epithelium of the sows uterus is a simple columnar type At the time of proestrus it has grown to a tall columnar, and pseudo stratified type which persists for about one week of the next cycle At diestrus vacuolar degeneration occurs and the epithelium is reduced again to a low columnar type During estrus the uterine muscle shows marked spontaneous activity A d a m s (1940) found that the myometrium of the sow responds to pituitrin with contractions and to epinephrine with relaxation during all stages of the estrus cycle

The epithelium of the vagina reaches its greatest thickness at estrus At this time there is a marked leukocytic infiltration of the mucous membrane, and leukocytes appear in the discharge during late estrus and early metestrus There is no actual cornification of the vaginal epithelium which begins to desquamate during estrus and reaches its lowest point of development during metestrus At the time of estrus the vulva is swollen pinkish in color, and its membranes are quite moist In late estrus and early metestrus a whitish discharge (sometimes referred to as the whites) containing mucus and large

numbers of leukocytes frequently collects on the vulva

Ovarian and pituitary hormones Estrogenic substances are found in urine at a level of 1 000 to 3,000 Mouse Units (M U) per liter during the first 19 to 30 days of pregnancy They then fall to a low level or disappear entirely until about the 70th day, when they reappear Following this they rise to a level of 5,000 to 10 000 M U per liter just before parturition After parturition they disappear rapidly (Roth *et al*, 1941)

West and Fevold (1940) studied the gonadotropic hormone content of the pituitary glands of various species of animals Sheep pituitary glands were found to contain the largest amount of leutenizing hormone (LH) and cattle pituitaries the least Pituitaries from swine showed great variation in LH content but in all instances the content was less than in sheep The hog pituitary contains larger amounts of follicle stimulating hormone than that of either the sheep or cow Assays of pituitary glands from female pigs indicate that the gonadotropic hormone level is lowest during the period of estrus and remains low until the eighth day of the cycle when it suddenly increases and remains high until the 21st day (Robinson and Nalbandov, 1951) There is a direct correlation between the rise or fall in the gonadotropic hormone content of the pituitary and the rise or fall in the number but not in the size of ovarian follicles Smith and Dortzbach (1929) determined that gonadotropic substances first make their appearance in the pituitary of pig fetuses when they reach a crown rump length of 17–18 cm, but significant amounts are present only after the fetuses reach the 20–21 cm stage

During the follicular phase of the estrus cycle no relaxin is found in the ovary of the gilt or sow, but during the luteal phase the content increases to 2.5–5 0 Guinea Pig Units (G P U) per gram of tissue During pregnancy the amount increases rapidly and reaches a maximal

concentration of about 10,000 G P U per gram by the time the fetuses reach a length of 12 5–15 0 cm The blood of the sow at mid pregnancy contains 2 0 G P U of relaxin per ml while the placenta con tains 0 5–2 5 G P U per gram The lack of relaxin in the follicular fluid and ovary of the sow in proestrus indicates that the corpora lutea are important in the forma tion of the substance (Hisaw and Zarrow, 1948) Injection of 5,000–9,000 G P U of relaxin three times daily for 4 days in gilts which have previously been given di ethylstilbestrol (5 mg daily for 7 to 10 days) results in a dilatation of the cervical canal and an edema of the vulva Associ ated with these changes the tissues of the cervix, vulva, and uterus show an increased water content (Zarrow et al, 1954)

Pregnancy Early observations (Ander son, 1927) indicated that ova remain in the Fallopian tubes for a period of 3 days before migrating to the uterus, but more recent work by Pomeroy (1955) shows that they reach the uterus within 48 to 72 hours Warwick (1926) demonstrated that intrauterine migration of ova takes place frequently in the sow and ordinarily the number of fetuses tends to become equal ized in the two horns when there is un equal production of ova on the two sides

The length of gestation varies somewhat with the breed of sow, and more work is needed to establish critical times for the various breeds In general, the average gestation period is 113 days with a range of 110 to 116 days These figures are in good agreement with the findings of Braude et al (1955), who studied a herd of Large White pigs over a 16 year period and found an average gestation period of 114 days with a range of 109 to 120 days No difference was observed in the length of the period for gilts or sows Later studies by Perry (1956) on Large White pigs in dicate an average period of 114 17 days and a range of 104 to 126 days for this breed These studies indicated that there is no direct relationship between the size of the litter or the age of the sow to the

length of the gestation period Although the sex of the fetus influences the length of pregnancy in some animals, this does not appear to be the case in pigs (McKeown and MacMahon, 1956) In the cow and horse, and possibly in the sheep and camel pregnancy is longer for male fetuses than for females, while the reverse is true for man and the guinea pig Data collected for the pig indicate that the mean dura tion of pregnancy for litters with a major ity of males is 114 6 days, while for litters with a majority of females the average time is 114 0 days

According to Warnick et al (1951), there is a steady rise in the number of eggs ovulated with each succeeding heat period from the first to the fourth heat, and gilts conceiving at the third heat farrow an average of 1 4 more pigs than those at the first heat These observations have been confirmed by others (Stewart, 1945 Ro bertson et al, 1951, Braude et al, 1955)

Placenta The placenta of the pig is the diffuse, epitheliochorial type In this type the fetal membranes are in simple apposi tion with the maternal uterus and the fetal and maternal bloods are separated by six layers of tissue These are the fetal capil lary endothelium connective tissue, al lanto chorionic epithelium (trophoblast), uterine epithelium, connective tissue, and uterine capillary endothelium

Parturition Perry (1954) studied the act of parturition in sows and described the following sequence of events   The ends of the chorionic sacs rupture early in the process, and this is followed by release of fetuses from the membranes and their free passage along the course of the uterine horns During the passage the fetuses re tain their placental connection through the very extensible umbilical cord The ruptured membranes remain in close con tact with the endometrium and serve to lubricate the canal for the fetuses

Stillbirths Asdell and Willman (1941) reported that the percentage of stillborn pigs in a herd studied over a 5 year period was 6 6 per cent They found that the oc

currence of large litters and advancing age in sows were both associated with an increased birth mortality. Spring farrowings had nearly twice the mortality of fall farrowings. Many of the stillborn pigs showed the presence of disproportionate organ weights or some pathological change. In a later study Perry (1956) stated that there is tendency for litters that contain either *more* or *less* than the average number of pigs to have a higher per cent of stillbirths. The lowest number of stillbirths was found in litters of 11 or 12.

Reproductive System in the Male

Sexual development. Spermatozoa first appear in testicular tubules of pigs when the animals are 20 weeks old. At 25 weeks of age all boars show the presence of sperm. The testicles increase slightly in weight between 10 and 17 weeks of age and then undergo a rapid growth during the 17th and 20th weeks. Between the 20th and 40th weeks there is a fairly constant growth of the testicles. In relation to body weight the testes increase slowly in size until body weight reaches 80 to 85 pounds; beyond this point the increase in weight of the testes is more rapid. No important differences are found in the sexual development of the large- or small-type male pig (Phillips and Zeller, 1943).

Testicular hormones. The cryptorchid testicle in the pig contains only about one-half as much androgenic hormone as the scrotal testicle. Hanes and Hooker (1937) reported that scrotal testicles contain one Bird-Unit of hormone in each 27 to 38.7 gm of tissue while cryptorchid testicles contain one Bird-Unit in each 53.5 to 86.7 gm. Androgens (which can be assayed by the usual capon comb growth technic and by colorimetric methods have been found in the urine of boars In 48-hour urine samples Green *et al.* (1942) found an average total amount of 3 017 mg. androsterone equivalent.

Physical characteristics of semen. A general discussion of the physical and chemical characteristics of semen is found

in the monographs of Anderson (1945) and Mann (1954a). No attempt will be made here to discuss the problem of artificial insemination, but a detailed account may be found in the review by Perry (1952).

The physical characteristics of boar semen differ in several ways from that of the ram or bull (Glover and Mann, 1954) (1) The volume of boar semen per ejaculate is much greater than for the other two species. (2) The concentration of sperm in boar semen is very low, and the bulk of the ejaculate is composed, not of sperm, but of accessory gland secretions (3) Boar semen contains a gelatinous material which may represent more than one-half the total ejaculate.

The volume of a single ejaculate from boars ranges from 150 to 500 ml, with the most common value being 250 ml (Mann, 1954a). In rams the average volume of a single ejaculate is 1.0 ml and for bulls 4.0 ml. In a study on a group of eight Large White boars Wallace (1949) found that the mean total volume of samples collected weekly over a 32-week period varied from 134 ± 78 ml. to 439 ± 85 ml. It was concluded that weekly collections over a long period of time are not likely to result in a reduced semen volume in normal boars. Vasectomy does not influence the volume of semen but castration results in an immediate decrease. The subcutaneous implantation of diethylstilbestrol tablets in young boars (2 to 16 weeks old) results in a semen volume which remains consistently below normal, but when the estrogen is discontinued, the volume rises to a normal level (Wallace, 1949).

The average concentration of sperm in boar semen is 100,000 per cu. mm with a range of 25,000 to 300,000 (Mann, 1954a). This is a very low concentration compared to that found in the bull (average 1,000,-000 per cu. mm.) and the ram (average 3,000,000 per cu. mm.). Comparatively little is known about the morphology of boar sperm, but they can be readily distinguished from those of other animals by

the rather corpulent appearance of the head (Glover, 1955). Under storage conditions *in vitro* boar sperm fail to survive as well as bull or ram sperm (Glover and Mann, 1954).

The gel portion of semen is primarily a product of Cowper's glands and forms a fairly constant proportion of the ejaculate. Johari (1956) found that the gel fraction of boar semen makes up approximately 30 to 40 per cent of an ejaculate, the other 60 to 70 per cent being composed of the liquid portion. After vasectomy the gel content may be somewhat higher and following administration of diethylstilbestrol the consistency may change to a syrupy character (Wallace, 1949).

Chemical composition of semen. The chemical composition of boar semen shows a relatively high content of citric acid, ergothioneine, and inositol, and a comparatively low concentration of fructose (Glover and Mann, 1954).

Citric acid, which is formed by the seminal vesicles and prostate gland, may play some role in gel formation (Glover and Mann, 1954) and may also be concerned with maintaining the osmotic pressure of semen (Mann, 1954b). There is a direct relationship between the formation of citric acid in the male accessory organs *and the activity of testicular androgen.* Following castration citric acid gradually disappears from the accessory gland secretions but reappears following the injection of testosterone (Humphrey and Mann, 1948; Mann, 1954a). The average concentration of citric acid in boar semen is 141 mg. per 100 ml.; in the horse, ram, and bull the concentration is 50, 137, and 750 mg. per 100 ml., respectively (Mann, 1954a).

Fructose can be utilized by sperm as a source of metabolic energy for motility (Glover and Mann, 1954). The bull and goat show a very high concentration of fructose (average 1,000 mg. per 100 ml.) while in boar and stallion semen the average content is only 50 mg. per 100 ml.

Ergothioneine is capable of maintaining intact the sulfhydryl groups in sperm in

a reduced form and may thus function in preventing the loss of motility which would result from oxidation of the sulfhydryl groups (Glover and Mann, 1954). Ergothioneine is formed in the seminal vesicles and is found in boar semen in an exceptionally high concentration. Glover and Mann (1954) found that the average concentration in semen collected from a Large White boar was 13.49 mg. per 100 ml. Mann (1954a) states that the average concentration for boars is 17 mg. per 100 ml. of semen.

The concentration of inositol found in the seminal secretions of the boar greatly exceeds the amounts hitherto recorded for any other material of plant or animal origin (Mann, 1951). Inositol probably plays an important part in maintaining the osmotic pressure of semen (Glover and Mann, 1954).

The concentration of the various organic and inorganic components of semen from several domestic animals is given in Table 3.2.

Semen fractions. In the boar, ejaculation is a prolonged process, lasting up to five to ten minutes, and the ejaculate may be divided into three major fractions (Glover and Mann, 1954; Glover, 1955). These have been designated as the *"pre-sperm", "sperm-containing", and "post-sperm"* fractions. Collectively, these make up a complete ejaculatory "wave," and this may be succeeded by a second wave, also fractionated. The chemical and physical characteristics of the various fractions are shown in Table 3.3 and Figure 3.1.

The pre-sperm fraction of the first wave is often yellowish in color, probably due to the presence of urine. The boar has a large preputial pouch which often contains stale urine. This fraction is ejaculated during the first minute or so and is free from gel and contains only a very few sperm (Glover and Mann, 1954).

The sperm-containing fraction is ejaculated next in the course of the second or third minute. It contains the bulk of the sperm, but only a little gel. In appearance it is a thick, creamy fluid.

The post-sperm fraction may be subdivided into a post-sperm *fluid* fraction and a post sperm *gel* fraction. The gel is thought to seal the cervix of the sow and prevent a backflow of semen.

Sperm transport. The observations of Mann et al. (1956) indicate that in the sow the sperm and seminal fluid reach the uterotubal junction at about the same time, shortly after mating. These investigators performed a laparotomy on a gilt 40 minutes after breeding and found that at this time the uterus presented a turgid appearance and showed peristaltic waves traveling upward in both horns Only a few sperm and no seminal fluid was found in the Fallopian tubes, but the rest of the reproductive tract contained sperm, seminal fluid, and seminal gel. In recent years there is more evidence that there are other important factors apart from inherent sperm motility which facilitate sperm transport in the female reproductive tract It has been observed that even dead sperm may rapidly reach the Fallopian tubes The evidence suggests that the transport of sperm is made possible not so much by the sperm's inherent motility as by contractions of the uterus, probably elicited by the release of oxytocin at the time of coitus (Mann et al., 1956). Rowson (1955) determined that in the cow it required ap proximately five minutes for a radio opaque oil to reach the tip of the uterine

TABLE 3 2

SPECIES DIFFERENCES IN THE CHEMICAL COMPOSITION OF SEMEN*

	Man	Bull	Ram	Boar	Stallion
Dry weight	8,200 (5,600–10,900)	9,530	14,820	4,600 (2,200–6,200)	2,450
Chloride.	155 (100–203)	371 (309–433)	87	328 (258–428)	264 (86–443)
Sodium ..	281 (240–319)	109 (57–201)	103	646 (280–837)	68
Potassium	89 (66–107)	288 (150–415)	71	243 (83–382)	62
Calcium	25 (21–28)	34 (24–45)	9	5 (2–6)	20
Magnesium.	14	12	3	11 (5–14)	3
Inorganic phosphorus	11	9	12	2	17
Total nitrogen	913 (560–1,225)	756	875	613 (334–765)	167
Non-protein nitrogen	75 (53–107)	48	57	22	55
Urea	72	4	44	5	3
Uric acid	6	6	6	3	
Ammonia	2	2	2	1	1
Fructose	224 (91–520)	540 (280–770)	247	12 (5–25)	15 (9–45)
Lactic acid	35 (20–50)	29 (15–43)	36	27	15
Citric acid	376 (96–1,430)	720 (340–1,150)	137	141 (41–325)	50 (30–110)
Total phosphorus	112	82	357	66	19
Acid soluble phosphorus	57 (27 5–93 5)	33	171	24	
Lipid phosphorus	6	9	29	6	
CO₂-content	41–60	16	16	50	24

* Results are average values (range in brackets) expressed in mg /100 ml except for CO₂ content (ml /100 ml)

From Mann (1954a)
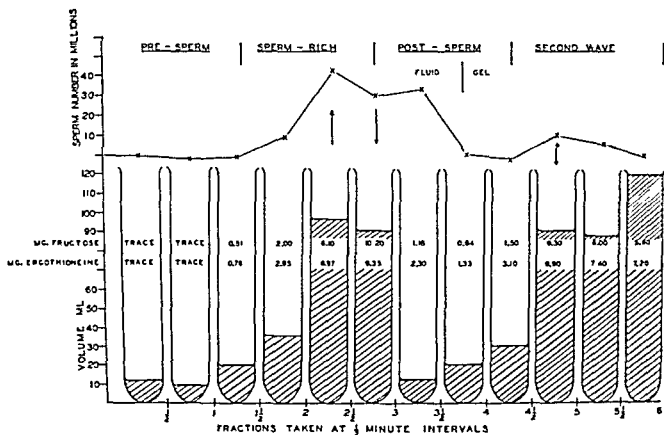
FIG. 3.1—Typical ejaculate from the boar, showing fractions taken at half-minute intervals, illustrating the volume, sperm, fructose, and ergothioneine contents. It may be noted that in the first wave the sperm content reaches its peak just before fructose and ergothioneine as emphasized by the arrows, but that there is considerable mixing of these fractions in the final ejaculate. (Glover, 1955.)

horn. After injection of oxytocin it required only two minutes for the oil to traverse the same distance. In these experiments it was concluded that the ascent of the oil through the reproductive tract was due to mechanical factors.

ENDOCRINE SYSTEM

Adrenal Gland

A variety of steroids have been isolated from the adrenal cortex of pigs (Dobriner et al., 1953). These include hydrocortisone, cortisone, corticosterone, and 11-dehydrocorticosterone.

Bruggemann et al. (1955) employed the level of 17-ketosteroids in urine as a measure of adrenal cortical function and found that the average excretion for young female pigs or castrated males was 3.9 mg. per day.

Anterior Pituitary

The anterior pituitary of pigs from fast-growing lines contains significantly more growth hormone per unit of tissue than does the same amount of tissue from slow-growing lines (Baird et al., 1952). Repeated daily doses of purified growth hormone to pigs result in a highly significant increase in the efficiency of food utilization (Turman and Andrews, 1955). Treated pigs show a hyperglycemia, and their carcass contains significantly more protein and moisture and less fat than that of untreated animals.

The anterior pituitary of gilts shows a progressively greater content of thyrotropic hormone up to the time the animals reach a weight of approximately 300 pounds and an age of 260 days. Gilts 32 days old (body weight 51 pounds) show an average of

TABLE 3 3

ANALYSIS OF BOAR SEMEN OBTAINED BY FRACTIONAL COLLECTION *

Fraction No	Time of Delivery from Urethra (min)	Characteristic Features	Volume (ml)	Sperm Concentration (thousands/μl)	Fructose (mg/100 ml)	Ergothioneine (mg/100 ml)	Citric Acid (mg/100 ml)	Chloride (mg/100 ml)	Inorganic Phosphate (mg P/100 ml)	Organic Acid Soluble Phosphate (mg P/100 ml)
1	0-1	' Pre sperm ' clear, slightly greyish fluid	46	Few sperm together with other cellular elements	2 9	6 3	31	360	2 4	32
2	1-4	"Sperm rich creamy fluid with little gel	100	327	4 5	12 4	50	350	1 3	25
3	4-7	'Post sperm fluid' with some gel	175	18	6 5	23 0	84	280	1 2	17
4	7 8	'Post sperm gel ' fraction completing the first ejaculatory wave	125	4	4 6	17 8	56	330	0 7	6
5	8-13	Fractions representing the second ejaculatory wave	140	88	5 5	21 7	69	340	1 6	26

* From Glover and Mann (1954)

44 Chick Units of thyrotropic hormone per gram of fresh anterior pituitary tissue, while at 260 days of age the hormone content has increased to 70 Chick Units per gram. Following this there is a gradual decrease so that at 436 days of age (body weight 520 pounds) the anterior pituitary contains 33 Chick Units of thyrotropic hormone per gram. Pigs of rapidly growing lines show a much higher level of the hormone in the pituitary gland than slow growing animals (Elijah and Turner, 1942).

Thyroid Gland

Iodine may first be detected in the thyroid gland of the fetal pig when the crown-rump length of the animal reaches 7–8 cm., corresponding to a fetal age of 46–50 days. At this time all of the iodine is in a water-soluble (inorganic) form. When the fetus reaches a crown-rump measurement of 8–9 cm. (age 52 days), thyroxine and diiodotyrosine may be found in the thyroid (Rankin, 1911).

There have been extensive trials on the use of various agents (e.g., thiourea, thiouracil) to depress thyroid activity in the pig and thereby hasten fattening. No attempt will be made to discuss the problem here, but a general review of the subject is found in the report of Blaxter et al. (1949).

METABOLISM

Carbohydrate Metabolism

Baby pigs appear to be highly susceptible to the development of an acute hypoglycemia immediately after birth, leading to the clinical syndrome known as "baby-pig disease" (Sampson et al., 1942). If pigs are fasted during the first 12 to 24 hours after birth, a severe hypoglycemia develops rapidly whereas they are more refractory to fasting at 120 to 140 hours of age, although the entire first week of life constitutes a critical period (Hanawalt and Sampson, 1947). Some have postulated that the regulatory mechanisms for gluconeogenesis are not well developed in very young pigs (Sampson et al., 1942). Several studies indicate that glucose is probably

the only sugar which can be readily utilized by newborn pigs (Newton and Sampson, 1951; Becker et al., 1954). When sucrose is used as the source of carbohydrate for baby pigs, the animals fail to show weight gains, develop diarrhea, and may die. Apparently newborn pigs lack the ability to hydrolyze the glycosidic bond of sucrose. In older mammals an intestinal sucrase brings this about. Sampson and Graham (1943) produced an experimental hypoglycemia in young pigs by the injection of insulin and found that if the animals were maintained in this state for more than 4 hours, they often failed to respond to even large doses of glucose. Pigs weighing 20 pounds which were fasted for 24 hours and then given 40 units of insulin often developed total blindness associated with a marked hypoglycemia and coma.

The form of the glucose tolerance curve obtained in pigs after intravenous administration closely parallels that seen in normal humans (Hanawalt et al., 1947). Although the horse, cow, and pig show considerable variation in their normal blood sugar values, all of these animals have a similar renal threshold for glucose (Link, 1953b). In the pig the threshold varies between 142.4 and 169 mg. of glucose per 100 ml. of blood (Fig. 3.2).

Fat Metabolism

Pigs on a fat-deficient diet show a variety of deficiency signs, including alopecia, a scaly, dandruff-like dermatitis of the tail, back, and shoulders, and necrotic areas of skin around the neck and shoulders (Witz and Beeson, 1951). The animals show an inhibition of sexual development and a significantly slower rate of growth than pigs receiving adequate amounts of fat in the diet.

When the diet of pigs contains an excessive amount of fish or fish meal their adipose tissue develops a greyish-yellow discoloration similar to that seen in "yellow-fat" disease in minks (Gorham et al., 1951). The discoloration is due to accumulation of an acid-fast pigment in the fat. Apparently two factors are necessary to produce this condition: excessive
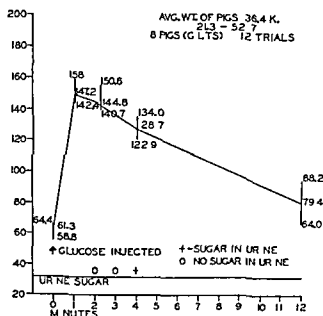
FIG 3 2—Renal glucose threshold for 8 unanesthetized pigs Urine collected from bladder (Link, 1953b)

amounts of unsaturated glycerides and in adequate amounts of tocopherols in the diet The administration of tocopherols results in a reduction of the acid fast pigment

ENVIRONMENTAL PHYSIOLOGY

The subject of environmental physiology of farm mammals has been reviewed by Findlay and Beakley (1954) Much of the literature deals with problems of heat regulation and the majority of observations have been made on cattle Data on the pig are scarce, but certain generalizations can be made for this animal

Body Temperature

The body temperature of the pig is not so stable as that of cattle or sheep This emphasizes the necessity of providing the means for swine to control their body temperature under extremes of the thermal environment (e g , shade and wallowing water in hot weather and artificial heat in cold weather) The marked variation in the body temperature of normal pigs is indicated by Pullar (1919), who studied rectal temperatures in a group of 83 animals and found a range of 97 2-106 6° F (average 102 15 ± 0 035° F) Palmer (1917), in some earlier studies reported a much narrower range of 101 6-103 6° F

It is well known that environmental temperature may exert a marked effect on body temperature of the pig In a controlled experiment using a psychrometric room Heitman and Hughes (1949) found that when the environmental temperature rose to 60-80° F the rectal temperature began to show a sharp increase The magnitude of the change was greater for heavier pigs (166-260 pounds) than for lighter animals (70-144 pounds) The same workers demonstrated the importance of water evaporation in controlling the body temperature of pigs When animals were kept in a dry room at 100° F their rectal temperature rose to an average of 106 8° F and all showed obvious signs of distress When water was poured on the floor and the pigs were allowed to roll in it the rectal temperature fell an average of 2° F within 90 minutes

In baby pigs the temperature regulating mechanism is not fully developed and they may show great fluctuations in their body temperature (Newland et al , 1952) During the first 30 minutes after birth the rectal temperature may drop as much as 3 to 13° F (Fig 3 3) The amount of the drop is related to the size of the pig and the environmental temperature Small pigs *(under 2 pounds) in a cold environment show a greater decrease than heavier animals* This is to be expected since small pigs have a greater surface area in proportion to body weight and must therefore produce more heat per unit of body weight than heavier pigs (Newland et al 1948) Under severe environmental conditions they are unable to do this The initial fall in body temperature is followed by a gradual return to normal which in environments of 60-75° F , may be reached in about 2 days and in environments approaching freezing in about 10 days Another interesting observation concerning the temperature regulating mechanism of baby pigs is that chilling results in a hemodilution This does not occur in adult swine and is the opposite of what is seen with a normally functioning temperature
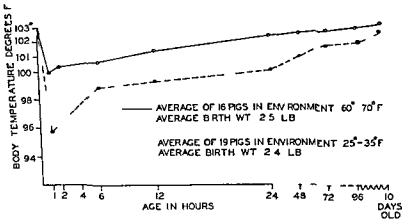
FIG 3 3—A comparison of body temperature drop in pigs raised in warm and cold environments (New land et al, 1952)

regulating mechanism (Newland *et al*, 1952)

Skin Temperature

The environmental temperature exerts an important influence on the skin temperature of pigs (Lee *et al*, 1941) When the air temperature is reduced from approximately 15° C to −12° C, the skin temperature decreases by an average of 14° C In contrast, sheep and goats are able to maintain a relatively constant skin temperature despite wide differences in the thermal environment

Other Effects of Thermal Environment

Feed consumption the rate of weight gain, and general behavior are affected by the environmental temperature At high environmental temperatures feed consumption decreases Light hogs (70–144 pounds) show the greatest gain in weight and the most efficient utilization of feed at an average environmental temperature of about 75° F, while for heavier hogs (166–260 pounds) the optimal temperature is approximately 60° F (Heitman and Hughes 1949) At higher temperatures (above 80° F) pigs become depressed and are reluctant to move about At lower temperatures (below 60° F) the animals often lie side by side and practice "community heating' (Heitman and Hughes, 1949)

Exposure of pigs to high environmental temperature results in a marked increase in breathing rate and a decrease in pulse rate (Fig 3 4) Tidwell and Fletcher (1951) determined that exposure of pigs

to direct sunlight for a period of 30 minutes resulted in an average increase in breathing rate of 81 per minute The effect of high temperatures on breathing rate varies with the breed and the weight of the pig Poland China pigs show a greater increase in rate than animals of the Duroc breed In lighter pigs (70–144 pounds) high air temperature has less effect on breathing rate than in heavier (166–260 pounds) pigs Animals of the heavier weight are unable to withstand temperatures much in excess of 100° F (Heitman and Hughes, 1949)
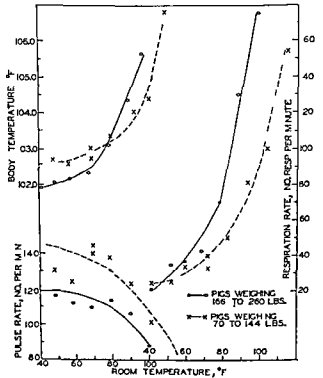
FIG 3 4—Effect of air temperature on body temperature and respiration and pulse rates of swine (Heitman and Hughes, 1949)

REFERENCES

Adams, E 1940 The reaction to pituitrin and adrenalin of the myometrium of the domestic sow Endocrinology 26 891

Allen, E., and Doisy, E A 1927 Ovarian and placental hormones Physiol Rev 7 600

Anderson, D 1927 The rate of passage of the mammalian ovum through the various portions of the Fallopian tube Amer Jour Physiol 82 557

Anderson, J 1915 The Semen of Animals and Its Use for Artificial Insemination Imperial Bureau of Animal Breeding and Genetics Technical Communication Edinburgh

Asdell, S A 1946 Patterns of Mammalian Reproduction Comstock Publishing Co., Ithaca N Y

——— and Willman, J P 1941 The causes of stillbirth in swine and an attempt to control it Jour Agr Res 63 345

Baird, D M Nalbandov, A V, and Norton, H W 1952 Some physiological causes of genetically different rates of growth in swine Jour Anim Sci 11 292

Baker, L N, Woehling, H L, Casida, L E And Grummer, R H 1953 Occurrence of estrus in sows following parturition Jour Anim Sci 12 33

Barber, R S Braude, R., and Mitchell, K G 1955 Studies on milk production of Large White pigs Jour Agr Sci 46 97

Barker, W L 1951 A cytochemical study of lipids in sows ovaries during the estrous cycle Endocrinology 48 772

Becker, D E, Ullrey, D E, Terrill S W And Notzold, R A 1954 Failure of the newborn pig to utilize dietary sucrose Science 120 345

Blaxter, K L Reineke, E P Crampton, E W, and Peterson, W E 1949 The role of thyroidal materials and of synthetic goitrogens in animal production and an appraisal of their practical use Jour Anim Sci 8 307

Bloor W R, Okey, R, and Corner, G W 1930 The relation of the lipids to physiological activity I The changes in the lipid content of the corpus lutem of the sow Jour Biol Chem 86 291

Bowland, J P, Grummer, R H, Phillips P H And Bohstedt, G 1949a The vitamin A and vitamin C content of sows colostrum and milk Jour Anim Sci 8 98

——— ———, and ——— 1949b The effect of the plane of nutrition on the composition of the sows colostrum and milk Jour Anim Sci 8 199

Braude, R., Clarke, P M, and Mitchell, K G 1955 Analysis of the breeding records of a herd of pigs Jour Agr Sci 45 19

———, Coates, M E., Henry, K M Kon, S K Rowland S J Thompson, S Y And Walker D M 1947 A study of the composition of sows milk Brit Jour Nutr 1 64

——— and Mitchell, K G 1950 Let down of milk in the sow Nature London 165 937

——— ——— 1952 Observations on the relationship between oxytocin and adrenaline in milk ejection in the sow Jour Endocrinology 8 238

Bruggemann, J Bonsch, K And Schmitt H 1953 Über den inneren und äusseren Stoffwechsel des gesunden und kranken Haustieres II Schilddrüsen und Nebennierenrinden funktion test an wachsenden Schweinen Zentralbl f Veterinärmed 1 63

Casida, L E 1935 Prepuberal development of the pig ovary and its relation to stimulation with gonadotropic hormones Anat Rec. 61 389

Cole, H H., and Hughes E H 1946 Induction of estrus in lactating sows with equine gonadotropin Jour Anim Sci 5 25

Corner, G W 1919 On the origin of the corpus lutem of the sow from both granulosa and theca interna Amer Jour Anat 26 117

Cort, J H, and McCance, R A 1953 The neural control of shivering in the pig Jour Physiol 120 115

Davis V E, Heidebrecht A A, MacVicar R, Ross O B., And Whitehair C K 1951 The composition of swine milk II Thiamine riboflavin niacin and pantothenic acid content Jour Nutr 44 17

Dorfman, K., Katzenellenbogen, E. R., And Schneider R 1951 Steroids from hog adrenal glands. Arch Biochem 48 167

Dukes, H H. 1955 The Physiology of Domestic Animals 7th ed Comstock Publishing Associates Ithaca N Y

———, and Schwartz, L H 1931a The blood pressure of the pig and the influence of non nervous and nervous factors on the cardiovascular apparatus Jour Amer Vet Med Assn 79 37

——— ——— 1931b On the nervous regulation of respiration in the pig Jour Amer Vet Med Assn 79 105

Friman, H D., and Turner C W 1942 The weight and thyrotropic hormone content of the anterior pituitary of swine Mo. Agr Exp Sta., Res Bull., No 357, p 27

Illenbecker, W., And Schiffert, A 1925 Der Harn und seine Absonderung In Lehrbuch der vergleichenden Physiologie der Haussäugetiere 3rd ed. Berlin

Findlay, J D., and Brakley, W R. 1954 Environmental physiology of farm animals In Recent Advances in the Physiology of Farm Animals Editor J Hammond Vol I, p 277 Butterworths Scientific Publications London

GLOVER, T 1955 The semen of the pig Vet Rec. 67 36

——, AND MANN, T 1954 On the composition of boar semen Jour Agr Sci 41 355

GORHAM, J R., BOE, N AND BAKER, G A 1951 Experimental yellow fat disease in pigs Cornell Vet 41 332

GRACE, V H MERRILL, C C. BUTZOW, R HINDREN, P AND SAMPSON, J 1951 Experimental uremia in young pigs Amer Jour Vet Res 12 206

GREEN, W W 1944 Urine secretion by boars. Amer Jour Vet Res 5 337

—— WINTERS L M RASH, J R , JR., AND DAILEY, D L 1942 The effect of sex on the development of the pig II Urinary excretion of androgens by boars of different lines of breeding Jour Anim Sci 1 111

HANAWALT, V M., LINK, R P., AND SAMPSON, J 1947 Intravenous carbohydrate tolerance tests in swine Proc. Soc. Exper Biol and Med 6 41

——, AND SAMPSON J 1947 Studies on baby pig mortality V Relationship between age and time of onset of acute hypoglycemia in fasting newborn pigs Amer Jour Vet Res 8 235

HANES F M AND HOOKER C W 1937 Hormone production in the undescended testis Proc Soc. Exper Biol and Med 35 549

HART, G H 1919 Endocrinology and its relation to reproduction Jour Amer Vet Med Assn 114 197

HEIDEBRECHT, A A., MacVICAR R ROSS, O B AND WHITEHAIR, C. K 1951 Composition of swine milk I Major constituents and carotene vitamin A and vitamin C Jour Nutr 44 43

HEITMAN, H JR., AND HUGHES E H 1949 The effects of air temperature and relative humidity on the physiological well being of swine Jour Anim Sci 8 171

HISAW, F L. AND ZARROW, M X 1948 Relaxin in the ovary of the domestic sow (Sus scrofa L) Proc. Soc. Exper Biol and Med 69 395

HOLMES, A D., TRIPPE, T., WOELFFER, E. A., AND SATTERFIELD, G H 1943 Ascorbic acid of cow s milk during four successive lactation periods Food Res 8 237

HUGHES E H AND HART, G H 1935 Production and composition of sows milk Jour Nutr 9 311

HUMPHREY, G F, AND MANN, T 1948 Citric acid in semen Nature London 161 352

JOHARI M P 1956 The gel mass in the semen of the boar Vet Rec. 68 158

KIMURA, G, AND CORNWELL, W S 1938 The progestin content of the corpus luteum of the sow (Sus scrofa) during successive stages of the oestrous cycle and pregnancy Amer Jour Physiol 123 471

LEE, R C, COLOVOS, N F., AND RITZMAN, E G 1941 Skin temperatures of pigs goats and sheep under winter conditions Jour Nutr 21 321

LINK, R P 1953a A study of the effect of repeated intraperitoneal infections of glucose in pigs Amer Jour Vet Res 14 150

—— 1953b Renal glucose threshold in the pig Amer Jour Vet Res 14 172

LUISADA, A., WEISZ, L. AND HANTMAN, H W 1944 A comparative study of electrocardiogram and heart sounds in common and domestic animals Cardiologia 7 63

MANN, T 1951 Inositol, a major constituent of the seminal vesicle secretion of the boar Nature London 168 1043

—— 1954a The Biochemistry of Semen Methuen and Co London

—— 1951b On the presence and role of inositol and certain other substances in the seminal vesicle secretion of the boar Proc. Roy Soc. 142 21

—— POLGE, C., AND ROWSON, L. E. A., 1956 Participation of seminal plasma during the passage of spermatozoa in the female reproductive tract of the pig and horse Jour Endocrinology 13 133

McKOWN, T, AND MacMAHON, B 1956 Sex differences in length of gestation in mammals Jour Endocrinology 13 309

NEIMEIER, K. 1911 Roentgenologische Beobachtungen am Magen Darmkanal des Schweines Jahresb Vet Med 68 6

NEWLAND H W., McMILLEN, W W., AND REINEKE, L. P., 1952. Temperature adaptation in the baby pig Jour Anim Sci 11 118

——, WALLACE D P., AND McMILLEN W S 1948 Some studies on temperature adaptation in the baby pig Jour Anim Sci 7 549

NEWTON, W C., AND SAMPSON, J 1951 Studies on baby pig mortality VII The effectiveness of certain sugars other than glucose in alleviating hypoglycemic coma in fasting newborn pigs Cornell Vet 41 377

PALMER, C. C. 1917 Effects of muscular exercise and the heat of the sun on the blood and body temperature of swine Jour Agr Res. 9 167

PERRIN, D R.. 1954 The composition of sow s milk during the course of lactation Jour Dairy Sci 21 55

—— 1955 The chemical composition of the colostrum and milk of the sow Jour Dairy Res. 22 103

PERRY, E J 1952 The Artificial Insemination of Farm Animals 2nd ed Rutgers Univ Press. New Brunswick N J

PERRY, J S 1954 Parturition in the pig Vet Rec 66 706

——— 1956 Observations on reproduction in a pedigree herd of Large White pigs Jour Agr Sci 47 332

PHILLIPS, R W, AND ZELLER, J H 1943 Sexual development in small and large types of swine Anat Rec 85 387

PLATNER, W S KIBLER, H H, AND BRODY, S 1948 Growth and development LXIV Electro cardiograms of mules, horses, cattle, sheep, swine and goats Mo Agr Exp Sta Res Bull No 419

POMEROY, R W 1955 Ovulation and the passage of the ova through the fallopian tubes in the pig Jour Agr Sci 45 327

PULLAR E M 1949 The rectal temperature in normal and infected pigs Brit Vet Jour 105 437

RANKIN, R M 1941 Changes in the content of iodine compounds and in histological structure of the thyroid gland of the pig during fetal life Anat Rec 80 123

ROBERTSON, G L, GRUMMER, R H, CASIDA, L E, AND CHAPMAN, A B 1951 Age at puberty and related phenomena in outbred Chester White and Poland China gilts Jour Anim Sci 10 647

ROBINSON, G E, AND NALBANDOV, A V 1951 Changes in the hormone content of swine pitui taries during the estrual cycle Jour Anim Sci 10 469

ROTH, S Y, MAYER, D T, AND BOGART, R 1941 Pregnancy diagnosis in swine by a chemical test Amer Jour Vet Res 2 436

ROWSON, L E 1955 The movement of radio opaque material in the bovine uterine tract Brit Vet Jour 111 334

SAMPSON, J, AND GRAHAM, R 1943 Studies on baby pig mortality III A note on experimental insulin hypoglycemia in the pig Jour Amer Vet Med Assn 102 176

———, HESTER, H R, AND GRAHAM, R 1942 Studies on baby pig mortality II Further obser vations on acute hypoglycemia in newly born pigs (so called baby pig disease) Jour Amer Vet Med Assn 100 33

SMITH, P E, AND DORTZBACH, C 1929 The first appearance in the anterior pituitary of the developing pig foetus of detectable amounts of the hormones stimulating ovarian ma turity and general body growth Anat Rec 43 277

SMITH, W W 1937 Swine Production The Macmillan Co New York

SPÖRRI, H 1954a Untersuchungen über die Systolen und Diastolendauer des Herzens bei den verschiedenen Haustierarten und ihre Bedeutung für die Klinik und Beurteilunglehre Schweiz Arch f Tierheilk 96 593

——— 1954b Untersuchungen ist dos Schwein fur den Herztod pradisponiert? Zentralb f Veterinärmed 1 799

STEWART H A 1945 An appraisal of factors affecting prolificacy in swine Jour Anim Sci 4 250

TIDWELL, A L, AND FLETCHER, J L 1951 The effect of summer environment on the body temperature and respiration rate of swine Jour Anim Sci 10 523

TRAUTMANN, A, AND ASHER, T 1940 Roentgenologische Untersuchungen zur Motorik des Colons des Schweines Arch wiss prakt Tierheilk 76 119

TURMAN, E J, AND ANDREWS, F N 1955 Some effects of purified anterior pituitary growth hormone on swine Jour Anim Sci 14 7

WALLACE, C 1949 The effects of castration and stilbestrol treatment on semen production of the boar Jour Endocrinology 6 205

WARNICK, A C, CASIDA, L E AND GRUMMER, R H 1950 The occurrence of oestrus and ovulation in postpartum sows Jour Anim Sci 9 66

———, WIGGINS, E L, CASIDA, L E., GRUMMER, R H, AND CHAPMAN, A B 1951 Variation in puberty phenomena in inbred gilts Jour Anim Sci 10 479

WARWICK, B L 1926 Intra uterine migration of ova in the sow Anat Rec 33 29

WEST, E., AND FEVOLD, H L 1940 A comparison of interstitial cell stimulating ovarian stimu lating and inhibiting actions of pituitary glands of different species Proc Soc Exper Biol and Med 41 446

WHITTLESTONE, W G 1954 The effect of adrenaline on the milk ejection response of the sow Jour Endocrinology 10 167

———, AND TURNER, C W 1952 Effect of acetylcholine on mammary gland of the lactating sow Proc. Soc. Exper Biol and Med 80 191

WIGGINS, E L, CASIDA L E AND GRUMMER, R H 1950 The effect of season of birth on sexual development in gilts Jour Anim Sci 9 277

WINTROBE, M M, ALCAYAGA R, HUMPHREYS S, AND FOLLIS R H. 1943 Electrocardiographic changes associated with thiamine deficiency in pigs Bull Johns Hopkins Hosp 73 169

WITZ, W M, AND BRESON, W M 1951 The physiological effects of a fat deficient d et on the pig Jour Anim Sci 10 112

ZARROW, M N, SIKES, D, AND NEHEN, G M 1951 Effect of relaxin on the uterine cervix and vulva of young castrated sows and heifers Amer Jour Physiol 179 687

VIRAL DISEASES

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Swine Influenza
(Flu, Hog Flu, Swine Flu)

Swine influenza is an acute, infectious respiratory disease of swine caused by the bacterium *Hemophilus influenzae suis* and the swine influenza virus acting in concert

HISTORY

The statement that no animal except man acquires influenza under natural conditions is encountered frequently in the older medical literature However, in the late summer or early autumn of 1918, a new epizootic disease, having many clinical and pathological similarities to influenza, appeared among swine in the Middle West This new disease was not a sporadic and localized outbreak, actually millions of swine became ill and thousands died during the first few months of its prevalence Its occurrence coincided with the greatest human plague of modern times — the 1918 pandemic of influenza — which killed about 21,000 000 of the world s people and caused illness in roughly twenty five times that many

According to Dorset et al (1922), Dr J S Koen, an inspector in the Division of Hog Cholera Control of the Bureau of Animal Industry, was the first to recognize the disease as being different from any previously encountered He was so much impressed by the coincidental prevalence of human influenza and by the resemblance of the signs and symptoms seen in man to those occurring at the time in hogs that he quite understandably became convinced

that the two diseases were actually the same He, therefore, gave the name of "flu" to this new disease of hogs The opinion of Koen that "flu' represented an entirely new swine epizootic disease, and that swine had been infected in the first instance from man was shared by some veterinarians and many farmers in the Middle West (Mc Bryde, 1927) Furthermore, the name ' hog flu or "swine flu" proved a generally ac cepted designation for the condition

Any claim that a disease is entirely new usually arouses justifiable skepticism How ever, the contention that a disease producing agent of one species has become estab lished in a new host and hence represents a new disease for that host does not par ticularly stretch one s credulity Such adaptations of infectious agents from one host to another have often been carried out experimentally in the laboratory, and there seems no reason why, under favor able conditions, such adaptations should not occur in nature The difficulty in the case of the influenzas of swine and man, when a proposal of their etiological identities was advanced, was that the causative agent of neither disease was known It was true however, as contended by Koen, that, allowing for certain differences be tween swine and man, swine and pandemic human influenza were indeed very much alike In both, fever, anorexia, cough, and other signs referable to the respiratory tract were prominent a leukopenia occurred in

both diseases, and, in both the degree of prostration was out of all proportion to the rest of the clinical picture In both diseases the onset was sudden, the course short, and convalescence slow but usually uneventful Both conditions appeared to be highly contagious Death, when it oc curred in either the human or the swine disease, was frequently the result of a water logged " bloody, edematous pneu monia It is, of course, evident that all of these similarities could have been a matter of chance and that one is not war ranted in drawing conclusions as to the relationship of two diseases on the basis of clinical and pathological resemblances alone Their etiological agents should be known and compared

It may be said in retrospect that Dr Koen was most uncannily correct in his speculations concerning the relationship between swine and human influenza, even without the benefit of knowledge concern ing the etiology of either disease All sub sequent work with swine and human in fluenza has supported the view that Koen s 1918 speculation concerning their relation ship was correct and that swine influenza did begin at that time as a new disease that hogs acquired from man The basis for this statement will be outlined in later sec tions of this chapter dealing with the etiol ogy and serology of swine influenza

Whether swine influenza of the type seen in the United States occurs in other parts of the world remains an open question A very closely related, if not identical, disease is seen occasionally in Northern Ireland (Lamont, 1938) and in England (Blake more and Gledhill, 1941) The viruses as sociated with these outbreaks, however, ap pear serologically to differ somewhat from the North American type and to be a little more closely related to current type A human strains (Glover and Andrewes, 1943) Gulrajani and Beveridge (1951) have clearly differentiated the virus of British swine influenza from a more prev alent one that causes infectious pneu monia of swine in England Almost cer tainly some of the respiratory ailments of swine described as occurring in European

countries and referred to as swine influenza are not indeed the same as the American disease Conditions referred to as influenza of swine have been described as occurring in Russia, Poland, the Balkans France, Germany, and the Scandinavian countries In some of these instances a virus having properties similar to that of swine influenza has been described as of etiological impor tance, and in some, an organism similar or identical to Hemophilus influenzae suis was encountered However, where actual comparisons have been made between European strains of viruses causing an in fluenza like disease in swine and American strains of the swine influenza virus the conclusion has been reached that the two agents are not identical Hjarre et al (1952) have extensively compared Amer ican swine influenza virus with that caus ing an enzootic virus pneumonia in Swed ish pigs and have concluded that the two agents are quite different Hjarre et al (1951) have recommended that the term 'swine influenza be applied only to those diseases with which a virus of the type found in North American swine influenza is associated, while respiratory diseases of swine, with which viruses other than the North American type of agent are associ ated should be referred to as 'influenza like" Review of the world literature sug gests that swine influenza, defined as recom mended by Hjarre and his co workers, is limited largely to the United States with sporadic outbreaks occurring occasionally in the British Isles Influenza like diseases in other parts of the world may, in all likelihood be instances of virus pneumonia of swine

ETIOLOGY

Swine influenza is a disease of complex etiology, being caused by infection with the bacterium H influenzae suis and the swine influenza virus acting in concert (Shope 1931b) H influenzae suis, the bacterial component of the etiological com plex (Lewis and Shope, 1931), is a small Gram negative, nonmotile hemoglobino philic bacterium showing a marked tend ency to pleomorphism especially in cul

tures that have been grown for longer than 24 hours In 24 hour cultures the pre dominant forms are small bacilli ranging from less than 0.5μ to 2μ in length and approximately 0.2μ in thickness In older cultures, long, threadlike forms are com mon, and these may form clumps of tangled masses of organisms Small coc coidal forms are also common in older cul tures The organism requires both factors V and X to support its growth on media It does not produce indol *H influenzae suis* is not pathogenic for swine when ad ministered alone in pure culture by way of the respiratory tract The organism is very similar, if not identical, to non indol producing, rough strains of *H influenzae* of human origin

The swine influenza virus, the other component of the etiological complex is pathogenic for white mice (Andrewes *et al*, 1934, Shope, 1935) and ferrets (Smith *et al*, 1933 Shope, 1934b), as well as for swine When administered to swine by way of the respiratory tract, it causes a tran sient mild illness, clinically quite distinct from swine influenza, which has been designated 'filtrate disease (Shope 1931b) Given intranasally to anesthetized ferrets the virus causes a febrile illness which may or may not result fatally, and produces a plum colored, edematous pneu monia of variable extent White mice to which the virus is given intranasally under ether anesthesia, regularly succumb to pneumonia The swine influenza virus is 80–120 mμ in diameter (Elford *et al*, 1936), a size identical with that of the type A human influenza virus Antigenically the swine influenza virus is closely related to the type A human influenza virus and in fact had it been initially isolated from man instead of swine, it would have been classed as a typical type A human influenza virus

Although *H influenzae suis* alone is not pathogenic and the virus alone is mildly pathogenic for swine the two when ad ministered together by intranasal instil lation cause in swine a clinically severe ill ness identical with swine influenza as seen in the field The disease caused by the two

agents acting in concert is highly conta gious and both agents transfer by contact from sick to normal animals (Shope 1934a)

The discovery of a hemoglobinophilic bacterium closely related to or identical with Pfeiffer s influenza bacillus in swine influenza was the first bit of scientific evi dence indicating the correctness of Koen s surmise of an actual relationship between human and swine influenza The discovery by Smith *et al* (1933) of a virus in human influenza very closely related to the one earlier discovered in swine influenza fur nished the second bit of scientific evidence supporting Koen s hypothesis of a relation ship between swine and human influenza

CLINICAL SIGNS

Swine influenza as it occurs in nature is not a disease of the individual rather it is a herd illness and proven single or sporadic cases do not occur It is essentially a disease of autumn and early winter and occurs annually among swine in the middle western states Its onset is sudden the morbidity rate in an infected herd is high, and practically all of the animals under one year of age become sick Fever and anorexia prostration of an extreme type cough and a peculiar abdominal type of respiration are salient features of the dis ease A leukopenia is usually to be ob served The period of illness is short vary ing from 2 to 6 days and in uncompli cated cases recovery is almost as sudden as the onset The mortality ranges from 1 to 4 per cent, but may be higher

Although the disease is a herd illness the observation of single animals under ex perimental conditions probably furnishes the clearest concept of the clinical picture The incubation period in swine infected by pen contact with influenza sick animals varies from 2 to 7 days with an average of 4 days Animals infected by intranasal in stillation with an infectious suspension containing swine influenza virus and *H influenzae suis* become ill more promptly and the incubation period in these cases is usually 24 to 48 hours Fever, that is a temperature of $101.0°$ F or higher, is in

all cases, the first observable evidence of illness Accompanying the rise in temperature, or following it very shortly, there is a mild degree of malaise, mild anorexia and a tendency for the animal to tire easily when made to exert itself While there is considerable variation in the temperature reaction as a rule on the first day it reaches 104 6° F, but seldom exceeds 105 8° F On the second day of illness, the fever, anorexia and malaise are more marked By the third day, the temperature as a rule has reached its peak and frequently exceeds 106 5 . F At this time, respiratory involvement is manifested by increased rate and a peculiar type of diaphragmatic breathing described popularly as thumping *Sometimes, at this stage, a paroxysmal type of cough is elicited when the animals are roused* They exhibit marked prostration refuse food, and lie listlessly in their pens Their condition on the fourth and fifth days is little altered from that on the third day Death may occur on the third, fourth fifth or sixth day and is frequently preceded by an exaggeration of all respiratory symptoms an increase in the prostration, and the onset of an active, incoordinated delirium during which the animal lies on its side *and makes running motions with its legs or, attempting to stand, staggers about the pen* On the sixth day, as a rule, the temperature has definitely receded in animals that are to recover and, from this time on recovery is rapid and ordinarily uneventful A leukopenia of moderate extent prevails throughout the course of illness (Shope, 1931a)

There are several features of swine influenza as a herd infection that perhaps should be mentioned in discussing the symptomatology of the disease, since they are more or less characteristic. Ordinarily there is a history that a period of wet, miserable, inclement weather, or some relatively minor change in swine husbandry, preceded the outbreak by several days Individual members of the drove do not sicken sequentially, but instead the illness gives the impression of starting explosively and spreading with great rapidity Ordinarily, within

a period of 24 hours, the majority of the animals show clinical evidence of illness As the herd illness progresses one gets the impression that the amount of prostration is out of all proportion to the other clinical signs that can be observed In a typical outbreak, the appearance of extreme illness is such as to indicate a very grave prognosis and suggests a death loss considerably higher than the 1 to 4 per cent usually encountered Recovery, when it does occur, affects the drove as a whole in much the same manner as did the onset and the majority of animals seem to recover almost simultaneously On about the fifth or sixth day of illness they will be found to be out of their nests and up and around again, and to show evidence of returning appetite Though still coughing and very noticeably gaunt in appearance most animals seem little the worse for their influenzal attack From this point on recovery of the drove is ordinarily rapid and uneventful although regain of the weight and condition lost during the period of illness is slow Furthermore, the troublesome postinfluenzal cough may last for three weeks or longer after recovery In view of the profound clinical illness shown by a typical case of swine influenza the sudden and rapid recovery seems almost miraculous As one writer has aptly recorded his impression in describing the clinical picture of the disease, The patient begins to recover about the time death is expected (Koen 1919)

GROSS PATHOLOGY

Necropsy of a swine sacrificed on the third or fourth day of illness with swine influenza reveals the following gross pathological picture The mucosa of the pharynx and larynx are as a rule mildly hyperemic and covered by a white glassy tenacious mucus The same type of exudate is present in moderate to copious amounts within the lumen of the trachea and the exudate may completely fill the lumen in the smaller bronchi and bronchioles In the small bronchi the exudate is of firmer consistency than higher in the respiratory tract, and not infrequently can
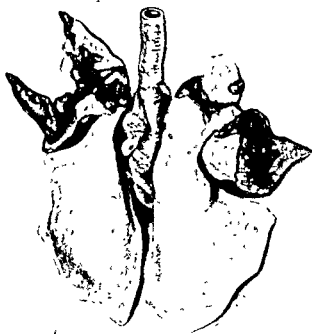
FIG. 4.1—Dorsal aspect of lung in experimental swine influenza to show typical appearance and distribution of atelectatic pneumonia. Lymph nodes at the hilum are swollen and edematous. Sharp demarcation of pulmonary lesions is noteworthy. Animal chloroformed on fourth day of illness. (Shope, Jour. Exper. Med.)
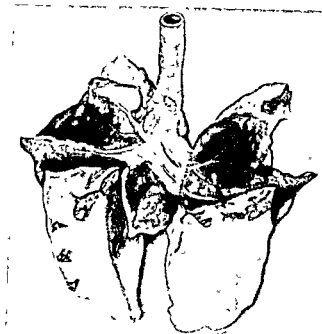
FIG. 4.2—Ventral aspect of same lung. (Shope, Jour. Exper. Med.)

be removed in small, white, semitranslucent, sago-like masses. In uncomplicated cases, the pleural sacs are free of either excess fluid or fibrin. The lungs present very constant and characteristic changes. The involved lung tissue is a deep purplish-red in color, and the line of demarcation between normal and pathological lung substance is very definite. Palpation of the involved lung reveals that it is firm and leathery and does not crepitate. The elements of the bronchial tree can be palpated and give the impression of being thickened. On cut section the bronchioles protrude from the surface, and the lung substance itself has a purplish-red, "beefy," pasty appearance throughout. The gross picture is that of a massive atelectatic pneumonia, irregular both as to amount and distribution. It is usually limited to portions of the cephalic, cardiac, and azygos lobes, and not infrequently involves large portions of all five of these lobes. The involvement is usually bilateral and irregularly symmetrical. If, however, it tends to be unilateral, the right side is almost al-

ways predominantly involved. An illustration of a typical case is presented in Figures 4.1 and 4.2.

The cervical and mediastinal lymph nodes are extremely enlarged and very edematous. Those at the hilum of the lung are sometimes so large as to resemble grapes. On cut section, they are found to be soft and to ooze fluid. Ordinarily there are few other pathological changes encountered in uncomplicated cases of swine influenza. The liver, kidneys, and spleen are usually negative, and the only noteworthy alteration of the gastrointestinal tract is a relatively extreme congestion and hyperemia of the gastric mucosa along the greater curvature in the cardiac end of the stomach.

Fatal cases of swine influenza present a somewhat different gross pathological picture. In these the mucosa of the trachea and larger bronchi are moderately congested and covered with a thick, tenacious, and sometimes frothy and blood-tinged mucous exudate. The smaller bronchi contain a more fluid exudate, sometimes in copious amounts, and frequently blood-tinged. A sero-sanguineous pleural exudate is frequently encountered, and this exudate sometimes contains considerable fibrin. The

lungs themselves are voluminous and heavy, and mottled purplish-red in color. Palpation reveals that only the apical, azygos, and cardiac lobes are consolidated; that is, the true pneumonia is limited entirely to the portions of the lung which in uncomplicated swine influenza are ordinarily involved. The diaphragmatic lobes exhibit a hemorrhagic type of pulmonary edema which, in most instances, is extreme. The markings of the interlobular septa are widened by fluid, and the lobes, as a whole, have a glistening, swollen appearance. When they are cut across, there is an outpouring of a frothy, bloody fluid. The lymph nodes are enlarged and edematous in fatal cases, just as they are in the nonfatal ones, and the scanty pathology outside the respiratory tract is also about the same.

HISTOPATHOLOGY

In animals killed on the third or fourth day of illness with swine influenza, films of the bronchial exudate, stained with methylene blue, reveal a rather constant and characteristic picture. The predominant cell in the exudate is the polymorphonuclear leukocyte, and of these there are many. Not infrequently they contain engulfed organisms, usually small thin bacilli, but occasionally larger bacillary forms or cocci. There are moderate numbers of lymphocytes in the exudate and smaller numbers of desquamated epithelial cells. Lying between the cells of the exudate are large numbers of extremely thin, very faintly staining, hairlike structures, evidently broken-off cilia.

Lung sections cut in such a way as to include small bronchi and terminal bron-
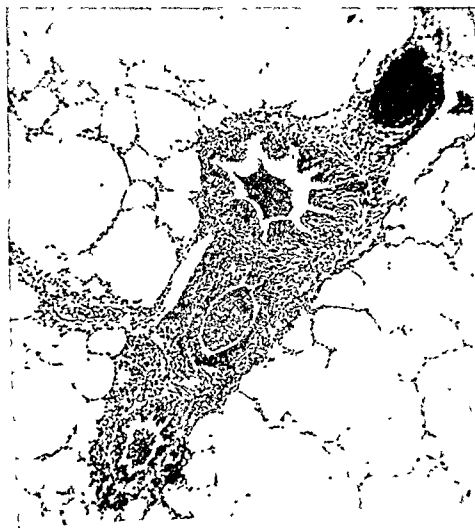

FIG 4.3—Section of lung from a spontaneous field case of swine influenza showing a bronchus in an area of compensatory emphysema and illustrating dense peribronchial round cell infiltration. Lumen of the bronchus contains a dense leukocytic exudate Eosin-methylene blue. X 75. (Shope, Jour. Exper. Med)

FIG. 4.4—Section of lung in experimental swine influenza showing dense leukocytic exudate in small bronchus and peribronchial infiltration largely with round cells. Animal killed on sixth day after inoculation. Eosin-methylene blue. X 75 (Shope, Jour. Exper. Med.)
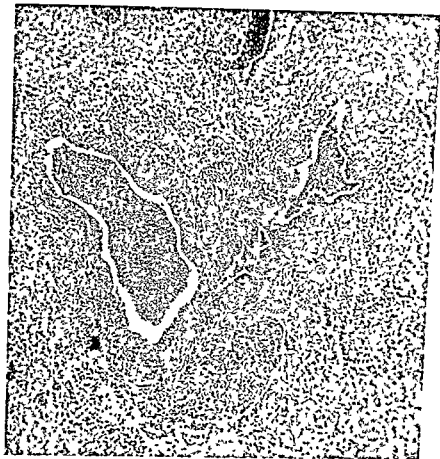
chioles, and including uninvolved and typically diseased lung, exhibit the following features: The small bronchi and terminal bronchioles are filled with a polymorphonuclear leukocytic exudate (Figs. 4.3 and 4.4). Bacteria are never numerous in this exudate and frequently they are not demonstrable or are present in such small numbers as to require careful search to find them. They are most numerous at the junction of the exudate and the bronchial epithelium. The cilia lining the smaller bronchi are either entirely gone or badly matted together. The lining epithelium is fragmented, in places partially desquamated, and the cytoplasm of many of the cells appears vacuolated. (Figs. 4.5 and 4.6). In the spaces created by the fragmentation of the lining epithelium, leukocytes, singly or in clumps, are sometimes seen (Fig. 4.6). There is an extensive peribronchial round cell infiltration (Figs. 4.3, 4.4, 4.5, 4.6). The areas of lung that appear grossly to be merely atelectatic are found histologically to present other

changes than atelectasis alone (Figs 4.7 and 4.8). They are of lobular distribution and sharply demarcated from adjacent uninvolved lung by interlobular septa, although a number of adjacent lobules may be and usually are involved. In these areas, the alveoli are collapsed and frequently contain desquamated cells, small numbers of mononuclear wandering cells, and occasionally some coagulated plasma. Leukocytes and red cells are not found regularly in the alveoli, although it is difficult to find sections, even from very early cases, in which the alveoli in some areas of the section do not contain leukocytes and occasionally red cells in small numbers. Leukocytes, when present, are most abundant in the alveoli opening directly into the terminal bronchioles. The alveolar walls are wrinkled and thickened and definitely infiltrated with mononuclear cells (Fig. 4.9). This infiltration is most marked in the alveolar walls adjacent to the bronchi, but is present and frequently conspicuous .... the entire area of atelectasis.
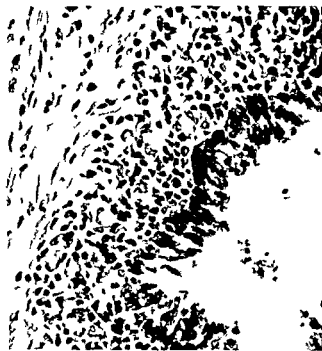
FIG 4 5—Section of a small bronchus from a spontaneous field case of swine influenza showing fragmented epithelium and extreme round cell infiltration of the submucosa Eosin methylene blue X 275 (Shope Jour Exper Med)
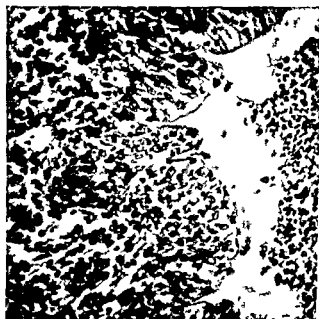
FIG 4 6—Section of a small bronchus in experimental swine influenza showing leukocytic bronchial exudate fragmented and vacuolated epithelium denuded of cilia and round cell infiltration of the submucosa Leukocytes have invaded the mucosa Animal killed on fifth day after inoculation Eosin methylene blue X 270 (Shope Jour Exper Med)

FIG 4 7—Section of lung in experimental swine influenza showing folding of pleura over an area of atelectatic pneumonia Animal killed on third day after inoculation Eosin methylene blue X 55 (Shope Jour Exper Med)

FIG 4 8—Section of lung from a spontaneous field case of swine influenza showing atelectasis with infiltration of the alveolar walls slight leukocytic exudate in some of the collapsed alveoli and compensatory emphysema Eosin methylene blue X 50 (Shope Jour Exper Med)

The interlobular septa are frequently widened owing not only to dilation of the lymph channels but to an apparent pull ing apart of the connective tissue elements and to some round cell infiltration The pleura overlying the atelectatic areas is sometimes wrinkled and thrown into small folds though unaltered otherwise (Fig 4 7) The lobules lying adjacent to areas of atelectasis are markedly emphysema tous exhibiting extremely thin alveolar walls many of them broken (Fig 4 8)

Histological examination of sections of pneumonic lung from fatal cases reveals the following findings The pleurae are usually overlaid with a network of fibrin in the meshes of which are myriads of leukocytes The bronchioles are completely filled with leukocytes their lining epithe lium is badly fragmented and partially desquamated and the bronchial walls themselves are densely infiltrated with round cells The alveoli throughout the section are filled with leukocytes red blood cells and coagulated plasma The alveolar walls are mildly folded thickened and in filtrated largely with round cells The lymph sinuses in the interlobular septa are dilated and contain leukocytes some lymphocytes and much lymph Sections of the edematous lower lobes of the lung re veal plasma filling the alveoli, which also contain small numbers of desquamated epithelial and red cells The cellular exu date in the bronchi is scant and there is much plasma The bronchial walls are thickened owing to intercellular edema and some round cell infiltration The pul monary capillaries are usually dilated and packed with red cells The lymph sinuses in the interlobular septa and beneath the pleura are widely dilated and contain in addition to plasma small accumulations of leukocytes and lymphocytes

DIAGNOSIS

Ordinarily the diagnosis of swine in fluenza occurring as a herd infection pre sents few difficulties The history of an outbreak of respiratory disease simulta neously involving most or all of the pigs in a drove in the late fall or early winter

FIG 4 9—Section of lung in experimental swine influenza showing thickening of alveolar walls in an area of atelectatic pneumonia Cells in filtrating the alveolar walls are largely round cells An mal killed on third day after inocula tion Eos n methylene blue X 270 (Shope Jour Exper Med)

is usually suggestive of swine influenza Examination of individual animals in a drove will reveal high temperatures pros tration and signs of respiratory tract in volvement Hog cholera a condition with which swine influenza might be confused begins more insidiously does not progress as rapidly to the stage of prostration and tends to show less respiratory tract involve ment than does swine influenza Because of the marked differences in the pathologi cal pictures of cholera and influenza a necropsy of a typically sick animal from the drove will usually settle the issue In case of doubt a bacteriological and virological diagnosis is relatively easy to make and will establish the identity of a case of swine influenza The bacterium H influenzae suis can usually be grown in pure culture on chocolate agar or on blood agar slants if exudate from small terminal bronchioles in an affected portion of the lung is selected for culture The virus component of the etiological complex can be demon strated by the inoculation of white mice intranasally with a suspension of bronchial exudate or pneumonic lung from a case of swine influenza Mice are inoculated

intranasally, while under ether anesthesia, by a technique described by Shope (1935). The inoculated mice will usually appear rough and ill on the third or fourth day or may actually die of a virus pneumonia by this time. Necropsy of such animals reveals the presence of a characteristic virus pneumonia which is transmissible in series in white mice.

Swine recovered from swine influenza develop antibodies in their sera which are capable of neutralizing the swine influenza virus (Shope, 1931b and 1932; Rosenbusch and Shope, 1939). The neutralization test is carried out best in white mice. In conducting the test, known swine influenza virus is mixed with blood sera from swine suspected of having had swine influenza, and the mixture is administered intranasally to anesthetized white mice. A positive serum, one from a pig recovered from swine influenza, will protect the inoculated mice from infection with the swine influenza virus. Serum from a pig that has not undergone an attack of swine influenza will not neutralize the virus, and mice receiving such a mixture will succumb typically to an influenzal virus pneumonia.

The leukocyte count is not of great help in establishing a diagnosis of swine influenza. Though a leukopenia is present in swine influenza, it also occurs in hog cholera to a somewhat more profound extent; hence in the two diseases, which might be confused most readily with one another, a low white count would not differentiate. An extremely low leukocyte count would, of course, strongly suggest hog cholera since the leukopenia in this disease is ordinarily more profound than in swine influenza. Swine influenza may be confused at times with virus pneumonia of swine and may be differentiated from this condition by its more rapid onset, more rapid spread throughout the drove, and more acute course. The viruses causing these two conditions also are entirely different, and the swine influenza virus is easily differentiated on the basis of the mouse inoculation or the serum neutralization test.

TREATMENT

There is no known specific therapy for swine influenza, and medication is not indicated. Careful nursing is of the greatest importance, however. Bedding down the animals in a dry, non-drafty, warm place, and leaving them comfortably alone is probably the best procedure. Clean straw should be used. Dust has a deleterious effect since it further irritates the inflamed mucous membranes and aggravates the troublesome cough. Since most of the animals will have a pneumonia of greater or lesser extent, they should not be unduly handled or roused. Plenty of clean drinking water should be accessible at all times since, because the animals are febrile, they are apt to be thirsty. There will be loss of appetite during the course of the acute illness but the appetite will return rather suddenly upon clinical improvement. Some care should be exercised at this time not to feed too heavily and to resume full rations gradually.

The losses from swine influenza are threefold. The most important of these is the actual death loss which ordinarily amounts to 1 to 4 per cent, but may go higher if the sick drove is poorly handled or if intercurrent complications enter the picture. The second source of loss is the shrinkage that growing or fattening hogs undergo during the course of their illness. This may involve from 1 to 4 weeks of feeding time. Both the death loss and the loss from shrinkage can be minimized by careful and intelligent nursing of the sick drove. The third loss inflicted by an outbreak of swine influenza is variable, but it does seem established that in some cases it may be sizable; this loss results from abortions or premature births due to an attack of the disease or from the farrowing of pigs that shortly after birth become weak and unthrifty and die. This source of loss can be avoided or minimized, in areas where influenza is of annual occurrence, by selecting breeding gilts that have recovered from the disease or by delaying the breeding dates until after the disease has passed

Young and Underdahl (1949a, 1949b 1950a 1950b) have studied extensively the matter of baby pig losses They have presented evidence to indicate that pigs born of gilts that undergo influenza virus infections during gestation suffer a reverse type of ana phylaxis frequently fatal, when they acquire swine influenza virus neutralizing antibodies on ingesting colostrum from their mothers soon after birth To judge from the figures presented by Young and Underdahl, these baby pig losses can be rather staggering among pigs farrowed in certain seasons of the year

IMMUNITY

Animals fully recovered from swine influenza are ordinarily immune to clinical reinfection and their blood sera contain antibodies capable of destroying the influenza virus Tests for these antibodies can be made in the laboratory in the so called virus neutralization test and their presence is sometimes used for diagnostic purposes Because of the fact that swine fully recovered from swine influenza are immune the history of two or more outbreaks in a given swine drove in a single season means either that one outbreak or the other was not true swine influenza Attacks following one another in rapid sequence can represent either a recrudescence of the original infection or the extension of pneumonia due perhaps to superinfection of the respiratory tract by secondary bacterial invaders The frequency with which hog cholera follows closely upon the heels of a respiratory infection which began as typical swine influenza is noteworthy and confusing

It is possible to immunize swine against influenza with a live virus vaccine administered subcutaneously or intramuscularly (Shope 1932 1936b), but the procedure is not without hazard of infection as was demonstrated during a small scale field trial There seems little doubt that, if there were a practical need for a vaccine one could be developed that would be both immunogenic and safe However, at the

present time no satisfactory swine influenza vaccine is commercially available and it seems doubtful were one available, that it would be used extensively

The immunology of the swine influenza virus is of further interest from a historical standpoint because through it information bearing on the origin of swine in fluenza has been provided As has been mentioned earlier, Koen was of the opinion that swine influenza began in 1918 at the time of the great human influenza pandemic and that swine had acquired their disease originally from man After the discovery of the swine influenza virus (Shope 1931b) and the human influenza virus (Smith et al, 1933), it was possible to compare these two agents and as has been pointed out earlier they proved to be very similar indeed from the standpoint of their pathogenic effects on experimental animals However the human virus was found to be slightly different serologically from the swine influenza virus This serological difference was not enough to necessitate classification of the two agents as different types of influenza virus (they are both classified as type A) but it was enough to enable their differentiation by serological tests Smith et al (1935) and Francis and Shope (1936) showed that the sera of animals recovered from infection with either the human or swine influenza virus would neutralize the homologous agent but usually not the heterologous agent Thus an immunological tool for differentiating the past influenza virus experiences of man appeared available A large series of blood sera were collected from people of different ages in England and the United States and these were tested for their ability to neutralize the swine and human viruses (Andrewes et al, 1935 Francis and Magill 1936 Shope 1936a) Much to everyone's surprise, it was found that the sera of practically all the adults of the study neutralized the swine influenza virus while the sera of almost all of the children failed to do so The findings were very similar for both the British and American sera and

appeared to bear no relationship to whether or not the individual supplying the sera also neutralized the human influenza virus For instance, though a number of the children's sera neutralized the human virus, almost none neutralized the swine virus, and conversely, though almost all adult sera neutralized swine virus many were devoid of neutralizing antibodies for the human agent It appeared from these findings that though almost all adults, to judge from their virus neutralizing antibodies had undergone a previous infection with a virus of the swine influenza type, none of the children had had a similar experience The fact that there was no evident relationship between the presence of human and swine virus antibodies indicated that the swine virus antibodies were probably specific and did not represent merely cross reactions from infection with the human agent So far as could be told from the limited number of sera falling in the age group between 10 and 19 years the critical age above which swine influenza virus neutralizing antibodies were almost always present and below which they were almost always absent was approximately 15 to 16 years Since the sera for the test had been collected in 1934 and 1935, it was very apparent that the period of time during which the agent responsible for the generation of these swine virus antibodies in human beings had been prevalent was suggestively near the year 1918 The conclusion seems inescapable that swine influenza virus and the pandemic human virus of 1918 were antigenically similar and that, as suggested by Koen on purely circumstantial evidence, swine may very well have acquired the agent, which we now know as the swine influenza virus, from man at that time A later study by Davenport et al (1953) with human sera collected in 1952 indicated that, in this series, swine influenza virus neutralizing antibodies were not detectible in sera from individuals under 29 years of age and were not found in relatively high titer until age 33 Here again, after a different time interval than that in the earlier studies,

the findings pointed to the likelihood that the antigenic stimulus responsible for the swine influenza virus neutralizing antibodies in human sera prevailed in the human population near and shortly following the year 1918 It would appear that the immunology of the swine influenza virus fits well with the historical aspects of the disease in indicating its origin as a new disease in 1918

EPIZOOTIOLOGY

It is probable that swine influenza has appeared in the Middle West each autumn since 1918 (Dorset et al, 1922, Dreher, 1922 McBryde, 1927, McBryde et al, 1928), although epizootics have varied in severity and extent from year to year Depending upon whether the onset of cold weather is early or late, the epizootics characteristically begin explosively in October or November The build up of cases is extremely rapid, and one gains the impression that the disease has arisen simultaneously at many different foci After the initial widespread outbreak, fresh swine droves are infected in smaller and smaller numbers until, by late December or early January as a rule, the epizootic appears to have run its course, and swine influenza disappears as a farm infection until the following October or November It seems certain that swine influenza, though highly contagious, would die out and disappear forever at the end of an outbreak if its causative virus did not possess some mechanism for lasting through the 9 month period during which the disease disappears as a farm infection Though the bacterium H influenzae suis can be found to persist indefinitely in the respiratory tracts of some recovered and apparently normal swine similar persistence of the virus has never been demonstrated

During this 9 month interval, nature has provided the virus with an ingenious mechanism for survival It has been found that the swine lungworm, a nematode parasitic in the bronchioles of the bases of the lungs of swine, is capable of harboring swine influenza virus and transmitting it from animal to animal (Shope, 1941)

Transmission of the virus by this intermediate host however is not quite so simple as the above statement might lead one to believe because the intermediate host has a required intermediate host of its own The swine lungworm is the actual carrier of the virus but since the lungworm must pass its first three developmental stages in the earthworm the latter becomes of importance as an accessory in the epidemiology of swine influenza

The life cycle of the lungworm as determined by the Hobmaiers (1929a 1929b) and by Schwartz and Alicata (1929 1931 1934) may be briefly summarized as follows The fully embryonated eggs are deposited by the adult female lungworm in the bronchi of the swine she infests These eggs are coughed up and swallowed and reach the outer world in the feces Their further development then depends upon their being ingested by earthworms Once within the earthworm the lungworm eggs hatch and the larvae develop to the third or infective larval stage usually becoming localized in the hearts, gizzard or calciferous gland of the earthworm intermediate host (Fig 4 10) They persist in this stage until their earthworm host is eaten by a pig In the pig the lungworms undergo two further developmental stages finally reaching the swine respiratory tract by way of the blood stream and lymphatics In the respiratory tract they become adults The whole of the cycle can occupy a span of several years for its completion or under the most favorable conditions can be completed in a little more than a month

Larvae developing from lungworm ova deposited during the time the host pig is undergoing an attack of swine influenza or even from those deposited for at least a short period after recovery are carriers of swine influenza virus (Shope 1941) A most puzzling feature of the transmission of swine influenza virus by the lungworm however is that virus cannot be detected by direct means either in the larvae in their earthworm intermediate hosts or in the adult lungworm after transmission to its definitive host the pig It appears to be

FIG 4 10—Third stage lungworm larvae as seen in a fresh press preparation of the calciferous gland of an experimentally infested earthworm X 69 (Shope Jour Exper Med)

present in an occult or masked form knowledge of its presence is furnished only by its subsequent behavior under very specialized conditions in the swine respiratory tract As a rule swine infested with lungworms that are carrying masked virus do not develop swine influenza immediately as might be expected Instead they remain to all outward appearances perfectly normal pigs However they are in a very precarious situation insofar as their eventual well being is concerned because all that is required to bring forth a severe or even fatal influenzal infection is the application of some stimulus of itself relatively harmless Several such provocative stimuli have been used but the one that has proved most regularly effective consists in the administration of multiple intramuscular injections of the bacterium H influenae suis These injections are ordinarily begun some weeks after the lungworm infestation has become established in the experimental swine and are spaced at intervals of 8 days Influenza usually follows the second or third injection A typical experiment is illustrated in Figure 4 11

Under natural conditions on the farm the stimulus responsible for provoking masked virus to infectivity appears to be meteorological in character and is associated with the onset of cold wet inclement weather It has been possible to duplicate
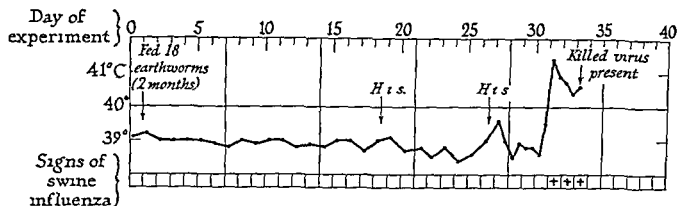
FIG 4 11—This pig was fed 18 earthworms which 2 months previously had ingested lung worm ova from swine with swine influenza Nineteen and 27 days later the pig was injected intramuscularly with H influenzae suis Four days after second inject on the animal developed swine influenza that was characteristic clinically and at necropsy Swine influenza virus was demonstrated in the respiratory tract (Shope Jour Exper Med)

this situation in the laboratory, using swine infected with masked virus and using adverse weather as the provocative stimulus (Shope 1955) Swine infested with lung worms known to be carrying masked swine influenza virus become ill in 2 to 4 days following some hours of exposure to inclement weather A typical experiment is illustrated in Fig 4 12

While little is known concerning the actual mechanism involved in the transmission of swine influenza by lungworms some facts indicating its applicability in the epizootiology of the disease are at hand It

has been found for instance that swine influenza virus can persist without giving any detectable evidence of its presence for at least as long as 32 months in third stage lungworm larvae in their intermediate hosts and for at least an additional 3 months in association with adult lung worms in the swine respiratory tract (Shope 1943a) This constitutes a total elapsed time of almost 3 years between the case of swine influenza originally supplying the virus and its eventual infection of the next pig This interval is roughly three times that which must be accounted
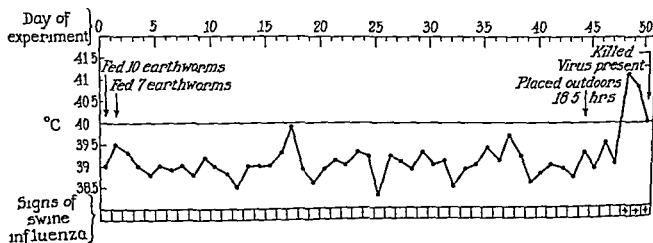
FIG 4 12—This pig was fed 17 earthworms containing third stage lungworm larvae carrying masked swine influenza virus Forty three days after this feeding it was exposed outdoors to inclement weather for 18 5 hours Four days later the animal came down with an illness that was typical clinically and at necropsy of swine influenza and swine influenza virus was demonstrated in its respiratory tract (Shope, Jour Exper Med)

for to explain the survival of the virus from one epizootic to the next

Another fact concerning the phenomenon seems to have a suggestive bearing upon the seasonal epidemicity of swine influenza Transmission of swine influenza virus by way of the lungworm takes place as described if the experiments are conducted between October and April that is during the fall winter, and spring months However experiments carried out from May through September have yielded negative results with only one exception The failure of the virus to cause infection by way of its intermediate host during the summer fits well with the known seasonal incidence of swine influenza under field conditions

Two types of field experiments have been carried out to demonstrate that lungworms carrying masked virus play the epidemiological role in nature that the experimental work described would indicate (Shope 1943b) In the first of these earth worms were obtained on Iowa farms that had a history of swine influenza as an annual occurrence After microscopic examination of these earthworms revealed their heavy infestation with lungworm larvae they were fed to experimental swine Later when a provocative stress was applied the experimental swine developed characteristic influenza Such experiments indicated that lungworms obtained under natural field conditions from premises where influenza was of annual occurrence were carriers of masked influenza virus and were hence a potential source of infection to any swine they infested

The other field experiment was conducted with swine which had acquired their infestations with lungworms presumably infected with masked influenza virus on their home farm in Iowa Three such animals were brought to my laboratory in New Jersey in September at a time when no clinically evident influenza existed among the swine on the Iowa farm from which the animals originated The swine were placed in isolation and kept under observation until November Then swine influenza virus infections were provoked by appropriate procedures in 2 of the 3 animals These 3 swine had been picked at random from a drove of about 50 shoats on the Iowa farm An assumption which seems warranted, in view of the fact that masked swine influenza virus had been demonstrated in lungworm larvae in earth worms from the same farm is that these swine were carriers of masked swine influenza virus when they were received at the laboratory and that this masked virus was in association with lungworms acquired on the home farm The swine in fluenza virus responsible for the disease developing in these animals is considered to be the same that would have caused them to sicken had they been left on the Iowa farm with their 50 whole and half brothers and sisters Since there is no reason for suspecting that the 3 swine chosen represented an unusual sample of the drove the finding that at least 2 were carriers of masked virus suggested an extremely high carrier rate for the drove as a whole

The finding of this high carrier rate suggests that the apparent paradox of swine influenza spreading throughout a drove and from farm to farm – faster than we realize that it can on the basis of the known incubation period – may not be paradoxical at all Instead of a swift spread of the virus throughout a drove and from drove to drove the field experiments suggest that it is probably widely seeded before the outbreak and then provoked almost simultaneously The great rapidity of spread therefore, is more apparent than real and represents a delusion resulting from the provocation of widely disseminated masked virus by a stimulus common to large geographical areas This wide spread provocative stimulus is in some way related to the wet cold changeable weather of late autumn It ordinarily prevails over much of the swine raising areas of the Middle West at about the same time, giving ample opportunity for mass provo-

cation of infection in large numbers of swine droves almost simultaneously Thus extensive outbreaks of swine influenza be gin in much the same manner and as a result of a stimulus similar to that which on a smaller scale can be demonstrated experimentally in the laboratory when swine known to be carriers of masked in fluenza virus are exposed for a few hours to wet cold inclement weather

A highly contagious disease like swine influenza is not thought of usually as re quiring the services of an intermediate host Ordinarily such a host is sought for or considered requisite only in those dis eases that do not transmit naturally by contact In the case of swine influenza however, one large period of the epidemio logic cycle – the interepizootic phase – becomes readily understandable only if the services of an intermediate host capable of maintaining the virus from one epizootic to the next can be invoked Once an epi zootic has gotten under way of course swine influenza spreads readily by contact just as does any other highly contagious disease

Theoretically, it should be possible to prevent and even eradicate swine influenza by preventing the exposure of swine to the intermediate host of the virus This would entail the prohibition of swine to all areas of ground containing earthworms infested with lungworm larvae From a practical standpoint it would necessitate the rearing of swine entirely on concrete On most midwestern farms this would not be feas ible

SWINE INFLUENZA AS A POSSIBLE HEALTH HAZARD TO MAN

Evidence has been presented in this chapter to indicate that swine influenza appeared as a new disease of hogs in 1918 and that it resulted directly from the infec tion of swine with the pandemic virus then widely prevalent in man If the swine virus had its origin from man in 1918 and ac tually stemmed from the lethal pandemic virus prevalent at that time in human beings it is quite natural to inquire con cerning its present potential to produce disease in human beings Does it at the present time present a possible health hazard to man? A direct answer to this question is not possible since no one to my knowledge has ever purposely at tempted to infect man with the swine in fluenza virus However the swine influ enza virus has now served as a disease producing agent in swine for roughly 38 years During all this time there is no conclusive evidence to indicate that it has ever reinfected man The serological evi dence obtained from study of human sera collected in 1934–35 and again in 1952 indicated that as has been pointed out earlier in this chapter the human proto type of swine influenza virus ceased in fecting man soon after the 1918 pandemic So far as can be determined from all the evidence at hand the swine influenza virus if indeed it was originally a human patho gen is now safely fixed in swine and oc curs as a natural cause of disease only in this species

REFERENCES

ANDREWES C H LAIDLAW P P AND SMITH W 1934 The susceptibility of mice to the viruses of human and swine influenza Lancet 2 859
—— AND —— 1935 Influenza observations on the recovery of virus from man and on the antibody content of human sera Brit Jour Exper Path 16 566
BLAKEMORE F AND GLEDHILL A W 1941 Discussion on swine influenza in the British Isles Proc Roy Soc Med 34 611
DAVENPORT F M HENNESSEY A V AND FRANCIS T JR 1953 Epidemiologic and immunologic significance of age distribution of antibody to antigenic variants of influenza virus Jour Exper Med 98 641
DORSET M McBRYDE C N AND NILES W B 1922 Remarks on hog flu Jour Amer Vet Med Assn 62 162
DREHER W H 1922 Swine diseases as we find them in the field Jour Amer Vet Med Assn 61 178

ELFORD, W J., ANDREWES, C H AND TANG, F F 1936 The sizes of the viruses of human and swine influenza as determined by ultrafiltration Brit Jour Exper Path 17 51

FRANCIS T , JR , AND MAGILL T P 1936 The incidence of neutralizing antibodies for human influenza virus in the serum of human individuals of different ages Jour Exper Med 63 655

—— AND SHOPE R E 1936 Neutralization tests with sera of convalescent or immunized animals and the viruses of swine and human influenza Jour Exper Med 63 645

GLOVER, R E , AND ANDREWES, C H 1943 The antigenic structure of British strains of swine influenza virus Jour Comp Path and Therap 53 329

GULRAJANI, T S , AND BEVERIDGE, W I B 1951 Studies on respiratory diseases of pigs IV Transmission of infectious pneumonia and its differentiation from swine influenza Jour Comp Path and Therap 61 118

HJARRE, A , DINTER, Z AND BAKOS, K 1951 Influenza och influensaliknande sjukdomar hos svin VI Nord Veterinarmotet p 7

—— —— —— 1952 Vergleichende Untersuchungen uber eine influenzaahnliche Schweinekrankheit in Schweden und Shopes Schweineinfluenza Nord Vet Med 4 1025

HOBMAIER, A AND HOBMAIER, M 1929a Die Entwicklung der Larve des Lungenwurmes Metastrongylus elongatus (Strongylus paradoxus) des Schweines und ihr Invasionsweg sowie vorlaufige Mitteilung uber die Entwicklung von Choerostrongylus brevivaginatus Munch tierarztl Wschr 80 365

—— AND —— 1929b Biologie von Choerostrongylus (Metastrongylus) pudendotectus (breut vaginatus) aus der Lunge des Schweines zugleich eine vorlaufige Mitteilung uber die Entwicklung der Gruppe Synthetocaulus unserer Haustiere Munch tierarztl Wschr 80 433

KOEN, J S 1919 A practical method for field diagnosis of swine diseases Amer Jour Vet Med 14 468

LAMONT, H G 1938 The problems of the practitioner in connection with the differential diagnosis and treatment of diseases of young pigs Vet Rec 50 1377

LEWIS, P A , AND SHOPE, R E 1931 Swine influenza II A hemophilic bacillus from the respiratory tract of infected swine Jour Exper Med 54 361

MCBRYDE C N 1927 Some observations on hog flu and its seasonal prevalence in Iowa Jour Amer Vet Med Assn 71 368

—— NILES, W B AND MOSKEY, H E 1928 Investigations on the transmission and etiology of hog flu Jour Amer Vet Med Assn 73 331

ROSENBUSCH C T , AND SHOPE R E 1939 The antibody response to swine influenza Jour Exper Med 69 499

SCHWARTZ, B AND ALICATA, J E 1929 The development of Metastrongylus elongatus and M pudendotectus in their intermediate hosts (abstract) Jour Parasit 16 105

—— AND —— 1931 Concerning the life history of lungworms of swine Jour Parasit 18 21

—— AND —— 1934 Life history of lungworms parasitic in swine U S D A Tech Bull 456

SHOPE R E 1931a Swine influenza I Experimental transmission and pathology Jour Exper Med 51 349

—— 1931b Swine influenza III Filtration experiments and etiology Jour Exper Med 51 373

—— 1932 Studies on immunity to swine influenza Jour Exper Med 56 575

—— 1931a Swine influenza V Studies on contagion Jour Exper Med 59 201

—— 1931b The infection of ferrets with swine influenza virus Jour Exper Med 60 49

—— 1935 The infection of mice with swine influenza virus Jour Exper Med 62 561

—— 1936a The incidence of neutralizing antibodies for swine influenza virus in the sera of human beings of different ages Jour Exper Med 63 669

—— 1936b Immunization experiments with swine influenza virus Jour Exper Med 64 47

—— 1941 The swine lungworm as a reservoir and intermediate host for swine influenza virus II The transmission of swine influenza virus by the swine lungworm Jour Exper Med 74 49

—— 1943a The swine lungworm as a reservoir and intermediate host for swine influenza virus III Factors influencing transmission of the virus and the provocation of influenza Jour Exper Med 77 111

—— 1943b The swine lungworm as a reservoir and intermediate host for swine influenza virus IV The demonstration of masked swine influenza virus in lungworm larvae and swine under natural conditions Jour Exper Med 77 127

—— 1955 The swine lungworm as a reservoir and intermediate host for swine influenza virus V Provocation of swine influenza by exposure of prepared swine to adverse weather Jour Exper Med 102 567

SMITH, W ANDREWES C H AND LAIDLAW P P 1933 A virus obtained from influenza patients Lancet 2 66

—— —— AND —— 1935 Influenza Experiments on the immunization of ferrets and mice Brit Jour Exper Path 16 291

YOUNG, G A., AND UNDERDAHL, N A   1949a Swine influenza as a possible factor in suckling pig
    mortalities I Seasonal occurrence in adult swine as indicated by hemagglutinin inhibitors
    in serum Cornell Vet 39 105
———— AND ————   1949b Swine influenza as a possible factor in suckling pig mortalities II
    Colostral transfer of hemagglutinin inhibitors for swine influenza virus from dam to off
    spring Cornell Vet 39 120
———— AND ————   1950a Swine influenza as a possible factor in suckling pig mortalities III
    *Effect of live virus vaccination of the dam against swine influenza on suckling pig mortal
    ities* Cornell Vet 40 21
———— AND ————   1950b *Swine influenza as a possible factor in suckling pig mortalities IV*
    Relationship of passive swine influenzal immunity in suckling pigs to rate of weight gain
    Cornell Vet 40 201

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CHAPTER 5

Virus Pneumonia of Pigs (VPP)

Virus pneumonia of pigs (VPP) quite likely is the world's most important swine disease. This statement is based on the high incidence of the disease, its worldwide distribution, and the estimated added cost to the producer. Economic loss because of VPP in England is estimated at $20,000 000 annually (Betts 1956). Losses in the United States are estimated at $120,000,000 annually (Young, 1956).

A chronic respiratory disease of swine has been described by many workers under different names but from the descriptions given, the diseases described are the same or at least very similar. Most common synonyms are infectious pneumonia of pigs (Pullar, 1948, Gulrajani and Beveridge, 1951), enzootic virus pneumonia (EVP) (Hjarre et al, 1952), infectious pig cough (Rislakki, 1953), and swine enzootic pneumonia (SEP) (Wesslén and Lannek, 1954) There are many reasons to suspect that the *Ferkelgrippe* of Kobe (1933) and some of the swine influenzas of Lamont (1938) and Blakemore and Gledhill (1941) also were VPP A good review of the possible relationships of these diseases has been written by Lamont (1952), although he does not include discussion of the infectious pneumonia of pigs by Pullar (1948, 1949 a b,c) The name *virus pneumonia of pigs* (VPP) presented by Betts (1952) has been accepted and is the name most characteristic, based

on his first identification of a specific virus etiology for this disease His work was an extension of studies reported by Gulrajani (1951 a b c) and by Gulrajani and Beveridge (1951)

The natural host for VPP is the pig No other natural hosts or experimental hosts have been found A possible exception is the adaptation of the VPP agent to tissue cultures of bovine embryonic skin and lung by Lannek and Wesslén (1955) Gulrajani and Beveridge (1951) found ferrets mice and embryonated hens' eggs insusceptible to VPP Similar observations on mice were made by Plowright (1953), Wesslén and Lannek (1954), and Fulton, *et al* (1953) The latter authors also found guinea pigs embryonated hens' eggs and Rhesus monkeys refractory to the VPP agent Embryonated hens' eggs were also found refractory to VPP by Penttinen and Rislakki (1953)

GEOGRAPHIC DISTRIBUTION

VPP probably has worldwide distribution, as judged from reports which apparently describe the same disease Pullar (1948) indicated occurrence of the disease throughout Australia Reports were made also from the United Kingdom by Gulrajani (1951), from Sweden by Hjarre *et al* (1952), from Finland by Rislakki (1953), from Canada by Fulton *et al* (1953) and Schofield (1956), and from the United

States by Beveridge (1953) Betts (1956)
Quin (1955), and Young (1956) The virus
pneumonia of pigs reported by Placidi and
Haag (1956) from Morocco is probably
not the same disease Both guinea pigs and
mice were infected by their agent which is
not characteristic of the disease described
by Gulrajani and Beveridge (1951) and
further identified as VPP by Betts (1952)

ETIOLOGY

The etiological agent for VPP is pre
sumed to be a filtrable virus of approxi
mately 250 mµ based on work by Betts
(1952) and Betts and Beveridge (1952)
No attempt has been made to classify this
virus generically The agent studied by Pul
lar (1948) did not pass a Seitz EK Special
filter The agent studied by Gulrajani and
Beveridge (1951) passed a Gradacol mem
brane with an A P D of 0 8µ but would
not pass a membrane with an A P D of 0 56
µ Infectivity of VPP was not destroyed by
storage at 20° C frozen or at 0° C in gly
cerol for 32 51 and 55 days (Gulrajani and
Beveridge, 1951)

CLINICAL SIGNS

The symptomatology of VPP has been
aptly described by Betts (1952) and is sum
marized in the following sentences VPP is
generally a chronic pneumonia with a high
herd morbidity and a low mortality Pigs
usually show first signs of the disease be
tween 3 and 10 weeks of age The incuba
tion period is from 10 to 16 days following
exposure A transient diarrhea may occur
for two or three days followed by a dry
nonproductive cough Suckling pigs may go
through a period of sneezing This symp
tom is not manifested by older pigs The
cough accompanying VPP is characteristic
and is most marked when pigs come out to
feed in the morning It may be elicited by
vigorous exercise Pigs may cough for only
one to three weeks or the coughing may per
sist indefinitely Respiratory movements re
main normal except in extreme cases In
general pigs retain their appetite but do
not grow well Loss of condition may be fol
lowed by markedly severe stunting Quite

often some of the pigs appear normal but
just grow slowly Apparent recovery from
VPP may be followed by a relapse or sec
ondary break down when pigs are about
16 weeks old

SPREAD OF INFECTION

VPP is spread from one pig to another by
direct contact or by inhalation of airborne
VPP virus Introduction of the disease into
a herd not previously infected can usually
be traced to the purchase of coughing feed
er pigs or asymptomatic adult carriers which
are added as new breeding stock As an ex
ample Pullar (1948) cites the spread of the
disease to 37 farms from a single consign
ment of sales yard pigs Young pigs usually
contract VPP from their mothers Herdwide
infection often occurs when pigs from sever
al litters are placed together for the first
time at weaning

INCIDENCE AND SEVERITY

There is much evidence that VPP is the
world's most prevalent swine disease La
mont (1938) in discussion of the disease in
the British Isles Germany Belgium and
the Balkan States stated that 60-70 per cent
of North Ireland pigs at bacon factories
show evidence of previous infection Pullar
(1948) indicated highest incidence of infec
tion among porkers He observed 68 per
cent infection among 152 feeder pigs An
thony is cited by Betts (1952), found evi
dence of respiratory infection in 61 per cent
of 1 000 pairs of lungs in pigs coming to
slaughter Betts (1952) found a similar pat
tern in 12 per cent of 1 000 lungs he exam
ined Although they give no specific figures
Fulton et al (1953) pointed out that pneu
monia in swine causes heavier losses in Sas
katchewan than do all other diseases Pneu
monia among swine in the United States
has been variably estimated by Beveridge
(1953) as 50 per cent in swine in Ohio by
Young and Underdahl (1955) as 50-70
per cent in midwestern swine and by
Betts (1956) as high as 71 per cent of a
single day's slaughter at Rochester New
York

A note of caution must be placed on

evaluation of the incidence of VPP based on the gross pathology alone Schofield (1956) in a histopathological study of pneumonias in Canadian pigs found respiratory infections in 50 of 75 herds or 67 per cent Material examined histopathologically disclosed only 29 of these 75 herds (39 per cent) had VPP Pattison (1956) similarly indicated that gross lesions regarded as typical VPP showed a wide variation histopathologically He concluded that there were several causes of these grossly similar lesions

PATHOLOGICAL CHANGES

The most common gross lesions in VPP are well demarked plum colored or grayish pneumonic areas in the apical and cardiac lobes of the lung Except for these areas the lung may have a normal appearance In the collapsed lungs as seen at necropsy there is no change in the level of the surface between the healthy and diseased portions These lesions are commonly referred to as atelectasis in a normal lung The high incidence of pneumonias in swine and their common lesions in the apical and cardiac lobes have led to acceptance of these lesions as normal in swine lungs A normal lung is portrayed in Figure 5 1 and is to be contrasted to an experimentally VPP infected lung as shown in Figure 5 2

Another typical lesion of VPP is enlargement of lymph glands involved in drainage of the lungs (Kobe 1934 Pullar 1948 Betts 1952 Wesslen and Lannek 1954 Pattison 1956 Schofield 1956) The pulmonary lymph glands contain virus

A variety of bacteria may be associated with VPP in a secondary category but contribute to the intensity of the disease and type of gross pathology (Kobe 1933 Pullar 1948 Gulrajani 1951 Betts 1952 Fulton et al 1953 Schofield 1956) Lungs free of bacteria but laden with virus are not uncommon VPP infections in absence of bacteria have been enhanced experimentally by migrating larvae of *Ascaris suum* (Underdahl and Kelley 1957)

Pattison (1956) found consistent histopathological changes in lungs from pigs infected experimentally with the MR strain of VPP This strain was isolated and described by Betts et al (1955b) and should represent the type species This agent caused extensive lymphoid hyperplasia of predominantly peribronchial peribronchiolar and perivascular distribution Schofield (1956) also used this lymphocytic infiltration together with hyperplasia of lymph nodes as a means of differentiating VPP from other pneumonias of swine It is of interest to note that histopathological

FIG 5 1 — Normal lung from 4 week old p g Note complete absence of atelectas s

FIG. 5.2 — Lung from 4-week-old pig experimentally infected with VPP virus 3 weeks before necropsy. Note atelectasis of cardiac (point of arrow) and apical lobes (lower right-hand corner).

lesions illustrated by Kobe (1931) as typical of *Ferkelgrippe* have the same characteristics as VPP lesions.

DIAGNOSIS

The diagnosis of VPP becomes a means of differentiating VPP from other pneumonias of swine. The primary disease from which it must be distinguished is swine influenza. These two diseases are distinct, so differential diagnosis is really not difficult, as already described by Pullar (1918) and Gulrajani and Beveridge (1951). Classical swine influenza is an acute disease with an incubation period of 2 to 4 days followed by elevated temperatures and extreme prostration (Shope, 1931a). By contrast, VPP is generally a chronic disease with an incubation period of 10 to 16 days. It causes only mild elevation in temperature, with little or no illness. The chronic, dry, nonproductive cough is also characteristic of VPP. Influenza is generally a seasonal disease being more prevalent in the fall and winter. VPP has no special seasonal occurrence.

Herd history and an appreciation of the incidence of the two diseases are also useful in differentiation. A chronic respiratory disease which has been a herd problem for months or even years is likely to be VPP.

The agent causing this disease may persist for months in the lungs of gilts or young boars held as breeding stock. Infection of young stock in the next generation may be from mother to suckling offspring. Such a situation can contribute to a perpetual chronic respiratory disease within a herd. By contrast, influenza virus only persists in lungs for a few days as an infectious entity. Infection is followed by immunity, and carrier animals rarely develop. Influenza must then recur in a herd by reinfection, which is unlikely until immunity wanes from the previous infection. Introduction of breeding stock is much less likely to cause an infection with swine influenza whereas VPP commonly follows the introduction of new stock. Chronic respiratory disease from a virus of the influenza group is unusual.

VPP has an unusually high incidence, as already indicated. In contrast, Young and Underdahl (1955) have shown in a five-year study of swine influenza among midwestern swine that the incidence is approximately 10 per cent. A large portion of this over-all incidence was contributed by a 21 per cent incidence in an epizootic year.

Differentiation between swine influenza and VPP is also possible by laboratory means (Shope, 1932, 1935, 1936; Young and Underdahl, 1950, 1955; Gulrajani, 1954

a b c) Influenza virus may be isolated by in oculation of mice or embryoned hens eggs and may be propagated continually in those hosts Egg propagated influenza virus will agglutinate red blood cells (RBC) of chick ens and several other species Antibodies de veloped by swine against the influenza vi ruses are capable of specifically inhibiting the agglutination of RBC Serum neutraliz ing antibodies also develop and are demon strable by inoculation of virus antibody mixture into embryoned hens eggs or mice

By contrast VPP has no other host than swine according to Gulrajani and Beve ridge (1951) Plowright (1953) Wesslen and Lannek (1954) and Fulton et al (1953) Further no demonstrable anti body develops following infection with VPP This characteristic has been re ported by Betts (1952) Betts and Beve ridge (1952) Hjarre et al (1952) Wes slén and Lannek (1954) and Betts (1956) VPP virus does not agglutinate chicken RBC according to Gulrajani and Beve ridge (1951) Betts and Beveridge (1952) Penttinen and Rislikki (1953) and Wes slén and Lannek (1954) Gulrajani and Beveridge (1951) and Hjarre et al (1952) were unable to demonstrate any serological relationship between swine influenza vi ruses and VPP by serum neutralization tests

TREATMENT

As with most diseases which have been relatively recently identified methods of treatment are controversial Since VPP does not cause stimulation of recognizable anti bodies a vaccine would appear to be use less Vaccines prepared by Pullar (1949) and by Schofield (1956) were of a bacterial nature and would be effective only against secondary invaders This relationship was recognized by Schofield and so stated Pul lar made no claim of effectiveness for his vaccine

Despite early claims of the in vitro sus ceptibility of VPP to broad spectrum anti biotics by Betts and Beveridge (1952) and

Wesslen and Lannek (1954) the test of time has demonstrated that VPP cannot be cured by antibiotic treatment It has been quite well established by work by Betts (1956) and by Lannek and Bornfors (1956) that tetracycline and oxytetracycline when given to pigs before they are exposed to VPP virus will prevent establishment of infection However the required doses of 2 to 4 grams of drug per pig per day are pro hibitive in cost The concept of Penny (1954) that VPP could be effectively treated with chloramphenicol was not supported by the data he presented Schofield (1956) indicated that antibiotics were useful to reduce the effects of secondary bacteria which caused an intensification of VPP Sul fonamides were found ineffective by Pullar (1949) and by Betts and Beveridge (1952) Penicillin and streptomycin have also been reported as useless in treatment of VPP by Betts and Beveridge (1952) and Betts (1956)

Good management can play an impor tant part in the control of disease Effects are minimized in well fed pigs in a warm dry environment free from drafts ac cording to Betts (1952) Underdahl and Kelley (1957) emphasize the importance of ascarid control as migration of the larvae through the lung intensify VPP

IMMUNITY

The failure of VPP infections to stimu late measurable antibody already has been discussed in the section on Diagnosis There is some evidence however that increased re sistance is bestowed upon herds and indi viduals as a result of exposure to VPP Betts (1952) indicates that VPP may be come acute with high morbidity and mor tality following introduction of the disease into a fully susceptible herd Greatest mor tality occurs among very young pigs Since this acute type VPP does not occur in herds in which the disease is enzootic some im munity or at least increased resistance is suggested Further the report by Macpher son and Shanks (1955) of only 6 per cent VPP in 670 old sows as compared to 53 per

cent among 1,000 gilts further substantiates the philosophy that some immunity is stimulated by VPP

CONTROL

VPP presents several unusual problems as far as disease control is concerned The fact that no appreciable immunity is developed precludes the use of a vaccine Chemotherapy, at least with present drugs is ruled out because the virus is not known to be affected *in vivo* by any drug Almost indefinite persistence of the virus in the lung up to at least 66 weeks reported by Betts (1952), presents a carrier problem by which the disease is passed from dam to offspring in succeeding generations The logical approach to control of such a disease would seem to be eradication followed by restricted movement of clean breeding stocks

The time tested principles of isolation and controlled rearing of breeding stock free from a specific disease have been used successfully to obtain VPP free herds of swine In the work with *Ferkelgrippe* in Germany Waldmann (1936) and Waldmann and Radtke (1937) began testing the usefulness of isolation of dams and their litters to control respiratory diseases of swine Macpherson and Shanks (1955) pointed out the advantage in using old sows because of the decreased incidence of

VPP in older animals as a source of new stock to be reared in isolation Using isolation principles Betts et al (1955a), Barber et al (1955) and Whittlestone and Betts (1955) succeeded in eradicating VPP from several herds of swine in England Their method of eradication consisted of the following stages (1) Farrowing sows in isolation to insure that any infection of the litter came only from the dam, (2) Determination of whether the litter was infected or not as judged by clinical examination supplemented by necropsy of one or more pigs per litter, if necessary and possible (3) Grouping of litters judged to be free from the disease but retaining the groups in isolation (4) Examination at slaughter of lungs from a considerable proportion of the group as a further check when they reached market weight (5) Replacement of the original breeding stock with the healthy progeny as soon as possible

Another method of obtaining breeding stock free of VPP and many other swine diseases has been described by Young et al (1955) and Young and Underdahl (1956) The same basic principles as already described are used except that the pigs are obtained originally from their dam by hysterectomy These pigs are raised in isolation on cows milk Details of this method are presented in Chapter 53

REFERENCES

BARBER R S BRAUDE R MITCHELL, K G AND BETTS A O 1955 The eradication of virus pneumonia from a herd of Large White pigs at a research station Vet Rec C7 690

BETTS A O 1952 Respiratory diseases of pigs V Some clinical and epidemiological aspects of virus pneumonia of pigs Vet Rec 64 283

—— 1951 *Ascaris lumbricoides* as a cause of pneumonia in pigs Vet Rec 66 749

—— 1956 VPP Virus pig pneumonia Jen Sal Jour 39 2

—— AND BEVERIDGE W I B 1952 Investigations on a virus pneumonia of long duration prevalent in pigs Jour Path and Bact 64 247

—— AND —— 1953 Virus pneumonia of pigs The effect of the disease upon growth and efficiency of food utilization Vet Rec 65 515

—— WHITTLESTONE J AND BEVERIDGE W I B 1955a Investigations on the control of virus pneumonia of pigs (V I P) in the field Vet Rec C7 (8)

—— —— TAYLOR J H AND CAMPBELL R C 1955b Virus pneumonia of pigs Further investigations on the effect of the disease upon growth rate and efficiency of food utilization Vet Rec 67 661

BEVERIDGE W I B 1953 Virus pneumonia of swine Vet Sci News. 7 13

BLAKEMORE F AND GLEDHILL, A W 1941 Some observations on an outbreak of swine influenza in England Vet Rec 53 297

BORNFORS S AND LANNEK N 1956 Treatment of enzootic pneumonia (Virus Pneumonia) in pigs with tetracycline Vet Rec 68 602

CARTER, G R, AND SCHRODER J D 1956 Virus pneumonia of pigs in Canada with special reference to the role of pleuropneumonia like organisms Cornell Vet 44 344

FULTON, J S, BURTON, A N, AND MILLAR, J L 1953 Virus pneumonia in swine Jour Amer Vet Med Assn 123 221

GULRAJANI, T S 1951a Studies on respiratory diseases of pigs I Modified haemagglutination inhibition technique for titration of influenza antibodies in pig and ferret sera Jour Comp Path and Therap 61 48

——— 1951b Studies on respiratory diseases of pigs II Antibody response to adjuvant vaccine against swine influenza Jour Comp Path and Therap 61 60

——— 1951c Studies on respiratory diseases of pigs III Persistence of influenza viruses in the respiratory tract Jour Comp Path and Therap 61 101

———, AND BEVERIDGE, W I B 1951 Studies on respiratory diseases of pigs IV Transmission of infectious pneumonia and its differentiation from swine influenza Jour Comp Path and Therap 61 118

HJARRE, A, DINTER, Z, AND BAKOS, K 1952 Vergleichende Untersuchungen uber eine influ enzaahnliche Schweinkrankheit in Schweden und Shopes Schweineinfluenza Nord Vet Med 4 1025

———, AND ——— 1954 Über Zuchtungsversuche mit dem Schweineinfluenza virus (Shope) und dem Virus der enzootischen Schweinepneumonie in vitro Nord Vet Med 6 610

KOBE, K 1933 Die Aetiologie der Ferkelgrippe (enzootische Pneumonie des Ferkels) Zentralbl f Bakt Parasit u Infekt 129 161

——— 1934 Die Ferkelgrippe Deutsch tierarztl Wschr 42 603

——— AND SCHMIDT, W 1934 Differentral Diagnose zwischen chronischer Schweinepest und Ferkelgrippe II Die Ferkelgrippe Deutsch tierarztl Wschr 42 163

LAMONT, H G 1938 The problems of the practitioner in connection with the differential diag nosis and treatment of the diseases of young pigs Vet Rec 50 1377

——— 1952 Virus pneumonia of pigs Vet Rec 64 442

LANNEK N, AND BORNFORS, S 1956 Prophylactic treatment of enzootic pneumonia (virus pneu monia) in pigs with tetracycline and oxytetracycline Vet Rec 68 53

———, AND WESSLEN, T 1955 Histological examinations of tissue cultures inoculated with a cytopathogenic agent from swine enzootic pneumonia Acta Path et Microbiol Scand 36 343

LEWIS, P A AND SHOPE, R E 1931 Swine influenza II A hemophilic bacillus from the res piratory tract of infected swine Jour Exper Med 54 361

MACPHERSON, R, AND SHANKS, P L 1955 The comparative incidence of pneumonia in sows and in bacon pigs with suggestions on the establishment of a pneumonia free herd Vet Rec 67 533

PATTISON, I H 1956 A histological study of a transmissible pneumonia of pigs characterized by extensive lymphoid hyperplasia Vet Rec 68 490

PENNY, R H C 1951 The treatment of virus pneumonia of the pig with chloramphenicol Vet Rec 66 730

PEVTTINEN, N AND RISLAKKI, V 1953 Om den i Finland forekommande smittosamma grishos tans etiologi 2 Adaptionsforsok i embryonerade honsagg och serologiska unders okningar Nord Vet Med 5 125

PLACIDI, L, AND HAAG, J 1956 La pneumonie à virus du porc—Etude clinique et experimentale d une epizootie au Maroc Recueil méd vét 132 5

PLOWRIGHT, W 1953 Observations on virus pneumonia of pigs in Kenya Vet Rec 65 313

PULLAR, E M 1948 Infectious pneumonia of pigs I General description differential diagnosis and epidemiology Australian Vet Jour 24 320

——— 1949a Infectious pneumonia of pigs II Morbidity, incidence, type, and location of le sions Australian Vet Jour 25 53

——— 1949b Infectious pneumonia of pigs III Transmission experiments and a field trial of a formolin killed vaccine Australian Vet Jour 25 123

——— 1949c Infectious pneumonia of pigs IV The relation of lung structure to lobe prefer ence Australian Vet Jour 25 262

QUIN, A H 1955 Problems of current interest in swine practice Vet Med 50 301

RISLAKKI, V 1953 Om den i Finland forekommande smittosamma grishostans etiologi 1 In fektionsforsok Nord Vet Med 5 113

ROSENBUSCH, C T, AND SHOPE, R E 1939 The antibody response to swine influenza Jour Exper Med 69 499

SCHOFIELD, F W 1956 Virus pneumonia like (VPP) lesions in the lungs of Canadian swine Canad Jour Comp Med 20 252

SHOPE, R E 1931a Swine influenza I Experimental transmission and pathology Jour Exper Med 54 349

——— 1931b Swine influenza III Filtration experiments and etiology Jour Exper Med 54 373

——— 1932 Studies on immunity to swine influenza Jour Exper Med 56 575

——— 1935 The infection of mice with swine influenza virus Jour Exper Med 62 561

SHOPE, R E 1936 Immunization experiments with swine influenza virus Jour Exper Med 64 47

UNDERDAHL, N R , AND KELLEY, G W , JR 1957 The enhancement of virus pneumonia of pigs by the migration of *Ascaris suum* larvae Jour Amer Vet Med Assn 130 173

WALDMANN, O 1936 Tagung der Fachtierärzte für die Bekämpfung der Aufzuchtkrankheiten Deutsch tierarztl Wschr 44 847

———, AND RADTKE, G 1937 Erster Bericht uber Erfolge der Bekämpfung der Ferkelgrippe durch die Riemser Einzelhuttenanlage Berl tierarztl Wschr 53 241

WESSLÉN, T, AND LANNEK, N 1954 The isolation and cultivation in tissue culture of a cyto pathogenic agent from pigs with enzootic pneumonia (so called virus pneumonia) Nord Vet Med 6 481

WHITTLESTONE, P, AND BETTS, A O 1955 The eradication of virus pneumonia of pigs from a commercial herd Vet Rec. 67 692

YOUNG, G A 1956 Is VPP a new swine disease? Norden News 30 6

———, AND UNDERDAHL, N R 1950 Neutralization and hemagglutination inhibition of swine influenza virus by serum from suckling swine and by milk from their dams Jour Immun 65 369

——— AND ——— 1955 An evaluation of influenza in midwestern swine Amer Jour Vet Res 16 545

——— AND ——— 1956 Measures to obtain and to maintain a heathy herd of livestock Jour Amer Soc Farm Managers and Rural Appraisers 20 63

——— ——— AND HINZ, R W 1955 Procurement of baby pigs by hysterectomy Amer Jour Vet Res 16 123

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CHAPTER 6

Transmissible Gastroenteritis

Transmissible gastroenteritis is an infectious, transmissible disease characterized by a high mortality in pigs less than a week to ten days of age. It affects swine of all ages but the mortality in older hogs is negligible. The disease was first reported in the United States by Doyle and Hutchings in 1946. Undoubtedly, the disease existed for many years before its specific nature was reported. It is now generally recognized as one of the important causes of death losses in young pigs.

Ordinarily it occurs sporadically, affecting swine on individual farms. Sometimes it affects many farms in a locality. It may occur at any time of year, but most of the outbreaks appear during the spring far rowing season. Spread of the disease is likely to be very rapid under conditions where litters of newborn pigs are close together, such as in a central farrowing house or where individual houses are assembled in a small area.

ETIOLOGY

Transmissible gastroenteritis is caused by a filtrable agent or virus. So far as is known, this agent does not cause disease in any species other than swine, although clinically similar diseases occur in several species, including humans in infancy. The virus soon dies out at room temperature and is only moderately resistant to common germicides such as phenol and formaldehyde. It remains active for many months when kept frozen in body tissues or gastrointestinal content. The virus is widely distributed in the bodies of infected pigs, being present in practically all tissues during the active stage of the disease. It seems to have a special affinity for epithelial tissue, particularly of the gastrointestinal tract. There is evidence indicating that the virus also multiplies in the respiratory tract. The virus is given off from the body, particularly in the bowel discharge, and very likely in other ways. It was found to persist for 8 weeks in an experimentally infected pig.

CLINICAL SIGNS

The clinical signs are suggested by the name, gastroenteritis. The incubation period may be as short as 12 to 18 hours. The disease usually spreads rapidly in swine of all ages affecting the whole herd within a few days. In older swine the symptoms are quite variable in severity. This is particularly true of brood sows. Some sows with affected litters do not show any noticeable symptoms, while others show inappetence, vomit, scour profusely, cease giving milk, and lose weight rapidly, rarely, a few die. The loss of weight in feeder hogs sometimes results in considerable economic loss to the owner. Instances have been seen in which the disease started in feeder hogs and then spread to the

young pigs Older swine usually show im provement by the end of 5 to 7 days and then recover

In young pigs, diarrhea is a constant sign Vomiting and polydipsia are other signs which occur frequently In young pigs the bowel discharge may be whitish yellowish, or greenish in color The in gested milk is often passed from the bowel only slightly changed There is usually rapid dehydration and loss of weight Evi dence of increased thirst in young pigs is indicated by a tendency to stand in or near the water supply and appear to be drinking Young pigs fatally affected usu ally die within 5 to 7 days after clinical signs first appear Some deaths occur as early as 48 hours The surviving animals are likely to remain stunted or unthrifty for some time The mortality may be nearly 100 per cent in pigs less than a week old The mortality is less if the pigs are older before they become infected

PATHOLOGICAL CHANGES

The principal gross lesions occur in the stomach and intestine Gross degenerative changes may be found in other organs especially in the kidneys and sometimes in the liver Emaciation and dehydration are evident in pigs that live for a few days after the onset Pigs that die early in the course of the disease have stomachs well filled with milk The lesions in the stomach and intestine vary greatly in degree There may be marked reddening of the fundal portion of the stomach and the greater portion of the intestine The mesenteric blood vessels may be engorged In many individual pigs there is no frank gross evi dence of inflammation of the stomach or intestine In these cases there is atony of the intestine, both large and small, but most commonly of the small gut The dis tended intestine is filled with liquid or semiliquid, giving the intestinal mass the appearance of being considerably increased in volume The dilated, liquid filled in testine is the most nearly constant lesion of the disease The kidneys usually show gross evidence of nephrosis, and urates may be found The medullary portion of the kidney is often congested The few older hogs that die of transmissible gastro enteritis nearly always show well marked gastrointestinal lesions

Microscopic examination does not show much more than is apparent on gross ex amination There is usually extensive loss of surface epithelium in the intestinal tract In addition to congestion and some hemorrhage, there is likely to be edema affecting particularly the intestinal villi, giving them a swollen appearance There is also more or less necrosis of intestinal epithelium, depending upon the severity and duration of the disease In pigs affec ted for about 4 days or longer, the necrosis may involve the epithelium to a consider able depth in the crypts The epithelial necrosis is usually patchy, and accumula tions of polymorphonuclear leukocytes are found near the necrotic areas There is usually more or less cellular infiltration of the intestinal wall, mostly by round cells Albuminous degenerative changes occur in the kidneys of most affected pigs The de generative changes in the liver are mostly fatty

DIAGNOSIS

The diagnosis of transmissible gastro enteritis can usually be made by observing the rapid spread of the disease and the high death rate among young pigs Profuse scouring and rapid spread among older swine followed by recovery starting in about 2 to 7 days make the diagnosis fairly certain Vomiting and scouring may occur in other infectious diseases such as hog cholera and in poisoning by corrosive or irritating substances such as arsenic In these latter conditions there are usually other distinguishing symptoms and lesions or toxicological findings which are peculiar to the specific disease A distinguishing feature of transmissible gastroenteritis is the readiness with which it can be trans mitted by contact or by feeding active material to susceptible newborn pigs

TREATMENT

No effective specific treatment has been found   Many different substances have been tried as treatments, none of which have proved definitely effective in con trolled trials   Some experimental treat ments possibly prolonged the survival time of infected young pigs, but none have significantly reduced the mortality   Spon taneous recovery, which is characteristic of the disease in older swine, may sometimes give an erroneously good impression of the effectiveness of treatment of mature ani mals

IMMUNITY

When sows that have lost their pigs from transmissible gastroenteritis are bred soon the next litters of pigs usually escape the disease   Pigs that are born a month or longer after an outbreak often live satis factorily   These observations suggest that resistance or protection eventually results from the sow being exposed to the disease This protection is apparently transmitted to the pigs mainly through the milk   The ability of the sow to transmit protection to the pigs probably lasts for less than a year   Serum from recovered animals can inacti vate the causative agent *in vitro*   However, such blood does not have significant pro tective action when given to young pigs

EPIZOOTIOLOGY AND CONTROL

The source of many outbreaks cannot be determined   In some instances it appears obvious that infection is brought in by visitors from infected herds or by moving machinery or other equipment from an in fected farm to an uninfected one   If a central source of feed or water becomes contaminated with the virus, extensive spread of the disease may result   The im portance of apparently healthy carriers of infection has not been determined   The fact that the disease is usually not a peren nial problem on a farm once infected, ex cept where there is continuous farrowing suggests that carriers are not common among swine

The control of an outbreak of transmis sible gastroenteritis is often impossible   In many instances all that can be done is to ride it out' until the outbreak is over and then breed the sows for the next farrowing   Early in an outbreak it may be possible to move the sows that are to farrow completely away from the infected places and thus save pigs that would other wise die   Continuous farrowing is not ad visable while the disease is present   Far rowing should be discontinued for about a month or longer in order to help control the disease   In some instances pregnant sows have been intentionally exposed to infection early in an outbreak in order to make use of whatever resistance or protec tion that develops   The sows are exposed by feeding them gastrointestinal tract and other viscera from infected pigs   It seems that about 15 days or longer is required for the sow to develop the ability to pro tect newborn pigs

REFERENCES

Bay W W 1952 Transmissible gastroenteritis in swine field herd studies Jour Amer Vet Med Assn 120 283
—— Doyle L P And Hutchins L M 1951 The pathology and symptomatology of trans missible gastroenteritis Amer Jour Vet Res 12 215
—— And —— 1953 Transmissible gastroenteritis in swine—A study of immunity Jour Amer Vet Med Assn 122 200
—— Hutchins L M Doyle L P And Bunnell D E 1949 Transmissible gastroenteritis in baby pigs Jour Amer Vet Med Assn 115 215
Cheever F S And Mueller J H 1947 Epidemic diarrheal disease of suckling mice I Manifestations epidemiology and attempts to transmit the disease Jour Exper Med 85 409
Doyle L P And Hutchins L M 1946 A transmissible gastroenteritis in pigs Jour Amer Vet Med Assn 108 257
Ewood F R And Whitthair C K 1955 The effect of transm ssible gastroenteritis on the metabolism of baby pigs Amer Jour Vet Res 16 116

FEENSTRA, E S, THORP, T, GRAY, M L, AND McMILLEN, W N 1948 Transmissible gastroenteritis of baby pigs Jour Amer Vet Med Assn 113 573

GORDON, I, INGRAHAM, H S, AND KORNS R F 1947 Transmission of epidemic gastroenteritis to human volunteers by oral administration of fecal filtrates Jour Exper Med 86 409

HAELTERMAN, E O 1956 Practical isolation equipment for baby pigs Amer Jour Vet Res 17 129

——— AND HUTCHINGS, L M 1956 Epidemic diarrheal disease of viral origin in newborn swine Ann New York Acad Sci 66 186

HAYS, M E 1955 Immunity to transmissible gastroenteritis in swine The Speculum Ohio State Vet Med Assn 9 11

HUTCHINGS L M 1947 Gastroenteritis in young pigs Vet Med 42 297

LEE, K M MORO, M, AND BAKER, J A 1954 Transmissible gastroenteritis in pigs Amer Jour Vet Res 15 364

NELSON, J M 1954 Control of transmissible gastroenteritis by inoculation with a field culture Jour Amer Vet Med Assn 124 387

PAPPENHEIMER, A M AND ENDERS J F 1947 An epidemic diarrheal disease of suckling mice Jour Exper Med 85 417

REBER, E F 1956 Airborne transmissible gastroenteritis Amer Jour Vet Res, 17 194

WHITEHAIR C K, GRUMMER, R H, PHILLIPS P H, BOHNSTEDT G AND McNUTT, S H 1948 Gastroenteritis in pigs Cornell Vet 38 23

YOUNG, G A HINZ, R W, AND UNDERDAHL, N R 1955 Some characteristics of transmissible gastroenteritis in disease free antibody devoid pigs Amer Jour Vet Res 16 529

———, UNDERDAHL, N R AND HINZ, R W 1953 A serum neutralization test for transmissible gastroenteritis of swine Cornell Vet 43 561
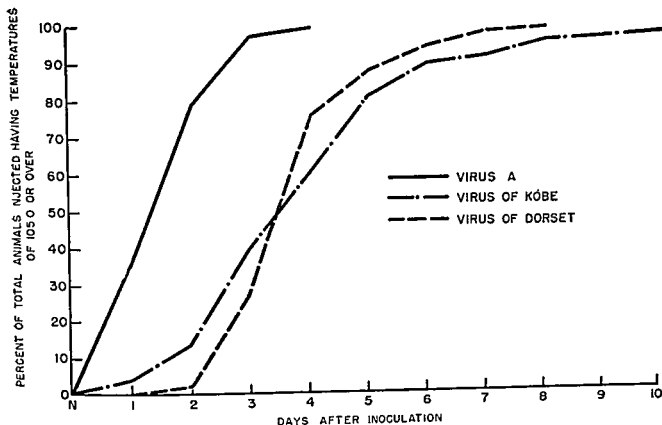
FIG 7 1—Virus A (Dunne et al 1952a) was a highly virulent variant causing a much earlier temperature rise than either the virus of Kobe and Schmidt (1934) or the virus of Dorset (1922) All animals were exposed to hog cholera virus by injection and not by contact

Like other viruses the virus of hog cholera is protein in nature but its chemical physical properties have not been fully investigated Electrophoretic tests show that it either carries a negative charge or is carried to the anode pole by other migrating proteins (Schwarte 1935)

CLINICAL SIGNS

A relative inactivity commonly termed slowness is one of the first external signs observed in hog cholera infections (Table 7 1) This is closely followed by a mild to marked anorexia characterized primarily by a decrease in food consumption Later the animal will come to the feeder eat lightly and then just nose around in the feed until he decides to go back to his resting place

If the animal's temperature is taken when the first inactivity is noticed, a definite fever will be in evidence Within two to six days after exposure to the virus the temperature of an infected animal usually rises above 104° F and may reach as high as 108° F (Fig 7 1) Although temperatures above 108° F have been recorded 106° F is more nearly representative of temperatures occurring during the course of the disease The peak of the temperature rise usually occurs between the fourth and eighth day of illness Concurrent with the temperature rise there is a corresponding drop in leukocyte count Total white cell counts of 9 000 to as low as 3 000 per cu mm of blood may be noted The lowest count usually occurs on the fourth to the seventh day after infection

Early in the course of the disease the eyes show a marked discharge which is associated with conjunctivitis This is readily observed in white pigs and may progress during the disease until the eyelids are completely adhered In some instances a moderate to severe nasal discharge is evident and in dry weather crusts may form

tween 1846 and 1855 only 93 outbreaks were reported. The disease proved to be a cyclic plague, causing extensive outbreaks in 1887, 1896, 1913, and 1926 (Quin, 1950).

Atherton (1923) estimated the hog cholera loss for the United States in animals alone from 1914 to 1924 to approximate 415 million dollars. Quin (1950) approximated the annual loss at 30 to 40 million dollars. The virus of hog cholera is still the cause of more swine deaths than any other infectious organism.

ETIOLOGY

The etiological agent causing hog cholera is a filtrable virus with the generic and species names of *Tortor suis*.

The causative agent was first thought to be a gram-negative bacterium (Salmon, 1899) which was eventually named *Salmonella choleraesuis*. In later investigations, De Schweinitz and Dorset (1903) showed the disease to be caused by a filtrable agent Confirmation and identification of the agent as a filtrable virus was made by Dorset *et al.* (1904).

The virus has a particle size of 22 to 30 mμ by direct measurements of electron micrographs (Reagan *et al.*, 1951) and is classified among the smaller of the filtrable agents. It appears to be spherical in shape.

The growth characteristics of the virus of hog cholera are limited to those shown by its propagation in tissue culture. Like other viruses, the causative agent of hog cholera cannot be grown in the absence of living cells. It has been propagated *in vitro* in various living cells from swine. Hecke (1932) first grew the hog cholera virus in swine tissues, using the Maitland and Maitland (1931) plasma clot technic. Boynton (1946) obtained virus propagation in cells from red bone marrow, serum, and modified Simms and Sanders saline solution. Frenkel *et al.* (1955) cultivated the virus in modified Tyrode's solution with suspended procine spleen tissue. Gustafson and Pomerat (1956) showed finely discernible cytopathological changes in splenic cells used to culture the virus Dunne *et al.* (1957a) demonstrated that hog cholera virus can be grown *in vitro* in leukocytes from peripheral blood.

TABLE 7.1

OCCURRENCE OF CLINICAL SIGNS OF HOG CHOLERA FROM THE DAY OF EXPOSURE TO THE VIRUS

Clinical Signs	Day of First Occurrence	Course
Decreased activity, "slowness"	2–6	Until death
Temperature rise	2–6	Until just before death
Leukopenia	2–6	May be intermittent until death
Exudative conjunctivitis	4–7	Until death
Huddling, piling	4–7	Until death
Vomition	4–8	Until death
Difficult respiration	4–8	Until death
Convulsions.	5–8	Seldom seen after 12 days
Constipation	5–8	Until death
Erythema	5–8	May become cyanotic before death
Diarrhea .	6–10	Intermittent until death
Weaving, incoordination	7–10	Until death
Hemorrhages of skin .	7–12	Until death
Cyanosis of skin	9–14	Until death
Blotching of ears	15–20	May be intermittent until death
Alopecia (partial) .	25–30	Until death
Death—peracute	.	4–7
Death—acute cases..		8–19
Death—subacute cases		20–29
Death—chronic cases		30–95

FIG. 7.3—A peculiar "blotching" of the ears, commonly associated with the more chronic type of hog cholera

high temperature or other metabolic disturbances cause a loss of bristles over much of the skin of the pig. Animals thus afflicted are almost certain to die.

On occasion, convulsions may be associated with hog cholera. This has been reported to be caused by a specific strain of virus, (Dunne *et al.* 1952a) but has been observed, though less commonly, in cases caused by other strains (Dimock, 1916; Brunschwiler, 1925). The clinical signs are characterized by a stiffening of the body, prostration, and violent running movements (Fig. 7.4). These actions may be sporadic or may occur sufficiently close

together to be considered continuous convulsions In all cases the animal appears to be in great pain at the time of the spasm. Although such signs of the central nervous system involvement are usually associated with early death, an animal may pass through a period of convulsive activity and live for many days.

Hog cholera is most commonly an acute disease with the course of infection terminating in death between 10 and 20 days after exposure to the virus. In peracute cases deaths may occur as early as 5 days The course may be extended beyond 20 days, in which case, the infection may be classified as more subacute than acute. Arbitrarily, cases lingering longer than 30 days can be called chronic. Swine have been known to survive for 95 days before dying with chronic hog cholera (Dunne *et al*, 1955). (See Table 7.1)

PATHOLOGICAL CHANGES
Pathogenesis

The virus of hog cholera is highly invasive as well as quite virulent. It apparently enters the body either through the upper digestive tract or through the respiratory system. Schwarte and Mathews (1954b) demonstrated that respiratory in-

FIG. 7.4—Late stages of a convulsion in a pig infected with hog cholera Running movements are beginning and the animal has a "wild look" in its eye

on the nose, severely impeding the passage of air.

Early in the disease and associated with the initial rise in temperature, infected animals become constipated and pass hard fecal pellets. Following a brief period of constipation a severe, watery, yellowish-gray diarrhea usually occurs. Often at this time, there is some evidence of vomition. Ascarids may be found in the vomitus and in the feces. Later, in uncomplicated cases, constipation may recur. In colder weather and sometimes even in hot weather the sick pigs will pile upon each other. This is particularly true just before the animals become moribund. During terminal stages of the disease sick pigs show a particularly noticeable weaving, staggering gait, which appears to be directly related to a weakness in the hind quarters. This is usually followed by a posterior paresis. Therefore, death is hastened by the inability of the animal to obtain water and food.

Active hyperemia of the skin develops relatively early in the disease and is quite noticeable in white hogs. This is usually concurrent with the initial temperature rise. A purplish discoloration, which extends over the abdomen, the snout, the ears, and the medial sides of the legs, occurs later in the disease near the terminal stages. A peculiar "blotching" effect on the ears may occur in acute infections but is more often seen in the more chronic cases (Fig. 7.3). In these instances, the discoloration may come and go as the animal alternately becomes more sick, shows improvement, and then inevitably relapses and dies. Chronically sick pigs often suffer from a partial alopecia characterized by a thinning of the bristles. Apparently the
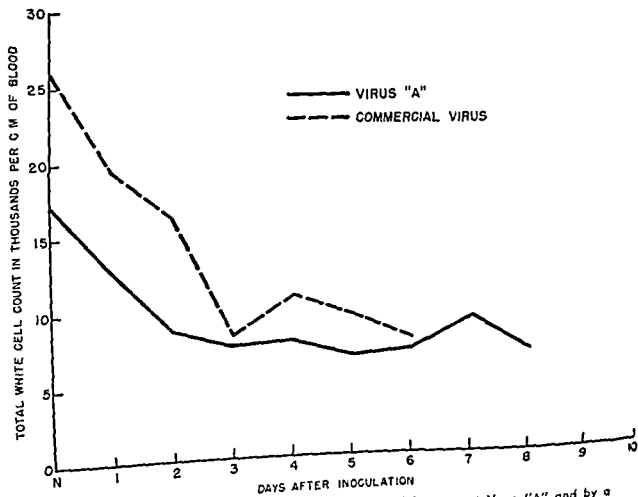

FIG. 7.2—The effects on total leukocyte counts produced by variant Virus "A" and by a commercially produced virulent virus are compared.

to almost black in color Other lymph nodes, notably the mesenteric and colic, are frequently characterized by peripheral hemorrhage On occasion, all lymph nodes may show peripheral hemorrhage Lymph nodes thus afflicted are described as mottled or "strawberry like" The intensity of hemorrhage varies with the type of disease Peracutely and chronically infected animals often die with little or no appearance of hemorrhage Acutely and subacutely infected animals usually manifest a relatively severe hemorrhage of the lymph nodes as well as other organs and tissues Secondary invasion by bacteria in all cases appears to increase the degree of hemorrhage which occurs

The tonsils of the pig like those of the human being are subject to a variety of infections, particularly those of a suppurative nature In hog cholera, however, there appears to be a necrosis of the tonsils which is believed by Maurer (1956) to be caused by infarction Experimentally, tonsillitis has been recorded in 38 per cent of 84 cases as shown in Table 7 3 The condition may appear early as a mild inflammatory reaction but develops later into a severe bilateral

TABLE 7 2

FREQUENCY OF OCCURRENCE OF GROSS LESIONS IN ORGANS AND TISSUES OF CHOLERA AFFECTED SWINE *

Organ and Tissue	Artificially Infected Series (Series A—48 Cases)		Naturally Infected Series (Series N—286 Cases)		Combined Series (Series A + N —334 Cases)	
	No Showing Lesions	Per Cent Showing Lesions	No Showing Lesions	Per Cent Showing Lesions	No Showing Lesions	Per Cent Showing Lesions
Kidney	46	95 8	263	91 9	309	92 5
Urinary bladder	46	95 8	232	77 6	278	83 2
Lymph nodes	44	91 6	235	82 1	279	83 5
Spleen	33	68 7	168	58 7	201	60 2
Larynx	32	66 6	167	58 3	199	59 6
Lungs						
Hyperemia	29	60 4	121	42 2	150	44 9
Hemorrhage	20	41 6	71	24 8	91	27 2
Inflammation	7	14 5	128	44 7	135	40 4
Large intestine						
Hemorrhage in mucosa	11	22 9	71	24 8	82	24 6
Hemorrhage in serosa	3	6 2	18	6 2	21	6 3
Inflammation	2	4 1	44	15 3	46	13 8
Heart	5	10 4	92	32 1	97	29 0
Liver	3	6 2	†	†		
Small intestine						
Hemorrhage in mucosa	2	4 1	29	10 1	31	9 3
Hemorrhage in serosa	4	8 3	20	6 9	24	7 2
Inflammation			1	3	1	3
Stomach						
Hemorrhage in mucosa	2	4 1	27	9 4	29	8 7
Hemorrhage in serosa	1	2 0	12	4 5	13	3 9
Inflammation	1	2 0	11	3 8	12	3 6
Skin ‡						
Cyanosis and hyperemia	10	20 8	64	22 4	84	25 1

* Kernkamp 1939a
† Insufficient data for comparative analysis
‡ Cyanosis and hyperemia in the skin are difficult to recognize in swine with pigmented skins therefore, the value of this statistic is not comparable with others in this table

fection was possible in controlled experiments. Under natural conditions, however, it is quite probable that most infections develop from exposure of the oral mucous membranes to virulent virus contained in infected food or water. It appears that the virus is capable of penetrating the unbroken membranes of the respiratory and tonsillar areas, and entering the blood stream. Penetration of the virus through the stomach or intestines has been shown to be improbable by the experiments of Dunne et al. (1957b), in which virulent blood virus was introduced into the stomach by means of a double capsule without producing hog cholera.

Evidence has been presented to show that leukocytes of the peripheral blood are capable of being infected and propagating the virus. Regional lymph nodes, examined microscopically, show an increased number of polymorphonuclear leukocytes within 5 hours following an intravenous injection of hog cholera virus, (Luedke and Dunne, 1957). Lymph nodes are the first tissues to show microscopic changes in the form of enlargement and hemorrhage Blood samples taken 15 minutes following an intravenous injection of the virus, have been shown to be infectious. At 30 minutes post-inoculation the blood was innocuous. Blood taken at 5, 8, and 13 hours after infection was also innocuous, but the samples taken at 16 and 18 hours produced hog cholera (Dunne and Luedke, 1957). It appears that at this time a high number of the cells of the reticular endothelial system are infected. The virus reaches a peak of concentration in the blood between 6 and 8 days after infection (Cole et al., 1946).

GROSS LESIONS

The pathological picture which is formed with the increasing concentration of virus is one of a septicemic disease characterized by petechial and ecchymotic hemorrhages. The circulation of the blood becomes slowed; blood vessels are weakened by hydropic degeneration of the endo-

thelial cells; and hemorrhages occur with varying intensity. In peracute cases it is often difficult to discern any evidence of hemorrhage in an infected animal. In cases with a somewhat longer course, however, an animal may demonstrate severe hemorrhages throughout its entire system. Hemorrhages are found most constantly in the kidneys and lymph nodes, occurring less constantly in the other organs and in the skin. The frequency of occurrence of hemorrhages and other lesions is shown in Tables 7.2 and 7.3 as observed by two separate investigations, Kernkamp (1939a) and Dunne et al. (1952a). Considerable variation in the percentage figures is noted. This difference may be associated with the susceptibility of the pigs, the strain (or source) of virus, the method of infection, the time of year, the conditions under which the sick animals were maintained, and, most importantly, the exposure to secondary infection. There is little doubt that secondary invaders intensify the lesions of hog cholera and even cause other lesions to appear.

Frequently, early in the disease, the skin is discolored by a marked erythema. As the disease progresses the erythema becomes cyanotic with the slowing of the blood. At times ecchymotic hemorrhages may occur on the medial sides of the legs, on the abdomen, and even extend up the sides of the animal. Secondary infection is believed to play an important part in the occurrence of some of these cutaneous, subcutaneous, and serous hemorrhages.

The lymph nodes ordinarily are the first tissues to show microscopic pathological changes, and are among the tissues which are the most constant in the development of lesions In the early stages of the disease, the lymph nodes appear to be somewhat enlarged and edematous. Usually there is evidence of hyperplasia, congestion, and hemorrhage. Lymph nodes including the parotid, submaxillary, cervical, bronchial, iliac, and superficial inguinal generally manifest a diffuse type of hemorrhage. These nodes may be moderately red

necrotic tonsillitis Sometimes the necrosis is aggravated by weed and wheat barbs which become lodged in the tonsillar crypts In many instances bacteria stimulate a suppurative reaction Primary suppurative tonsillitis does occur in the pig, but in hog cholera infected swine suppurative tonsillitis is more likely to be the result of a secondary bacterial invasion of diseased tissue

Hemorrhages of the epiglottis and larynx appear to vary with the conditions under which the disease is observed Hoskins (1916) recorded 75 4 per cent of 500 swine artificially inoculated with the virus of hog cholera as having some degree of laryngeal hemorrhage As shown in Table 7 2 the lesion was found in 60 2 per cent of 334 artificially and naturally infected swine Table 7 3 shows that laryngeal hemmorrhages of the epiglottis (and larynx) occurred in only 23 8 per cent of 84 experimentally produced cases More than 60 per cent of these were limited in nature

Approximately half of the pigs suffering from acute or subacute hog cholera exhibit some degree of acute bronchopneumonia or congestion of the lungs Under ideal experimental conditions this occurs less often Ecchymotic hemorrhages of the lungs are not uncommon but their frequency of occurrence appears to vary considerably with the conditions and complicating infections Pleuritis may be present as the result of a secondary invasion by bacteria Atelectasis and interstitial edema are only of minor importance as lesions of hog cholera

The heart is usually flabby, shows some myocardial congestion and, on infrequent occasions, coronary thrombosis Hydropericardium fibrinous pericarditis, pericardial hemorrhages and endocardial hemorrhages are more commonly associated with complicating bacterial infections

Lesions of the kidney occur more frequently in hog cholera than any other pathological change These may occur on the subcapsular surface of the kidney in the form of sparse petechial hemorrhages which because of their smallness and lack of numbers (sometimes as few as 2 or 3) may be difficult to detect In the other extreme they may occur as numerous ecchymotic, turkey egg hemorrhages ranging in size to 2 mm in diameter (Fig 7 5)


FIG 7 5—Severe 'turkey egg' ecchymotic hemorrhages of the kidney most often seen in complicated hog cholera infections

TABLE 7 3
SUMMARY OF THE GROSS LESIONS OF 84 PIGS EXPERIMENTALLY INFECTED WITH HOG CHOLERA *

Lesion	Mild	Moderate	Severe	Total	Per Cent of Total
Conjunctivitis	10	24	25	59	70 2
Erythema	11	9	5	25	29 8
Subcutaneous hemorrhage		1	1	2	2 4
Lymphatic peripheral hemorrhage	17	15	8	40	47 6
Lymphatic diffuse hemorrhage	12	33	15	60	71 4
Tonsillitis	11	13	8	32	38 1
Epiglottis—petechiation	13	5	2	20	23 8
Hydrothorax	5	9	0	14	16 8
Hemothorax	2	3	0	5	6 0
Hydroperitoneum	2	6	1	9	10 7
Thymus—petechiation			1	1	1 2
Hydropericardium	6	3	1	10	11 9
Epicardial petechiation	2	5	4	11	13 1
Myocardial degeneration	19	9	2	30	35 7
Coronary occlusion	1	2	0	3	3 6
Fibrinous pericarditis	0	3	1	4	4 8
Pleuritis	0	2	0	2	2 8
Bronchopneumonia	30	12	7	49	58 3
Pneumonic ecchymosis	1	5	7	13	15 7
Interstitial edema	0	0	1	1	1 9
Atelectasis	3	2	0	5	6 0
Peritonitis	1	2	0	3	3 6
Gastritis	16	13	9	38	45 2
Gastric edema	1	1	0	2	2 4
Gastric serosa petechiation	0	2	1	3	3 6
Gastric mucosa petechiation	1	4	2	7	8 3
Enteritis—small intestine	9	3	1	13	15 5
Severe intestinal serosal and mucosal petechiation	0	1	0	1	1 2
Colitis cecitis, diffuse necrotic	6	5	3	14	16 8
Acute colic ulceration	0	0	1	1	1 2
Button ulcers	7	5	5	17	20 2
Colonic petechiation	1	4	4	9	10 7
Nutritional enteritis	3	0	0	3	3 6
Acute catarrhal colitis	9	7	1	18	21 4
Hemorrhagic colitis	1		2	3	3 6
Splenic infarction	5	13	5	23	27 4
Renal cloudy swelling	9	3	2	14	16 8
Renal petechiation cortex	25	19	10	54	64 3
Renal cortex ecchymosis	3	0	10	13	15 5
Renal pyramids ecchymosis	8	7	9	24	28 6
Nephritis	0	2	0	2	2 4
Hepatic fatty metamorphosis	0	6	0	6	7 1
Cholecystic congestion	2	1	0	3	3 6
Cholecystic petechiation	3	3	3	9	10 7
Cholecystic ecchymosis	2	2	1	5	6 0
Hepatic scars	2	14	4	20	23 8
Ascariasis	2	6	3	11	13 1
Cystic petechiation	40	22	4	66	78 6
Cerebral congestion	15	31	15	61	72 6
Petechiation of omentum and mesentery	0	0	1	1	1 2

* Dunne *et al*, 1952a

FIG 7 7—Severe button ulcer formation in the colon of a pig experimentally infected with hog cholera
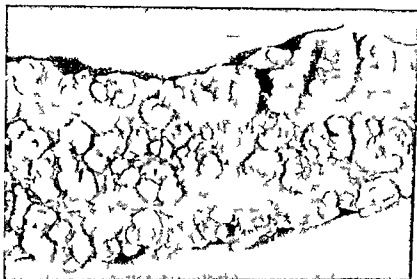
have concentric lines (Fig 7 7) Shown to be associated with small infarctions in the intestine (Dunne *et al*, 1952b), this lesion is believed to be diagnostic of hog cholera The lesion was shown to begin as a small indiscernible necrotic area to which small fecal plaques adhere (Fig 7 8) Later secondary infection sets in and the circular lesion progressively becomes larger as exuding mucus combined with cellular

and fecal debris becomes encrusted over the eroded surface

Although hog cholera virus cannot be given full credit for the generalized necrotic enteritis that is associated with the virus infection it does predispose the animal to bacterial infection Generalized necrotic enteritis is certain to complicate the pathological picture of hog cholera if the proper microorganisms are present and mucosal re

FIG 7 8—Early button ulcer formation showing beginning concentric lines

More frequently occurring than either of these extremes is the mildly to moderately petechiated kidney which is so characteristic of uncomplicated hog cholera (Fig. 7.6). While hemorrhages of the medullary portion of the kidney are less common than those on the surface of the cortical area, they do occur with relative frequency in the form of petechiae and sometimes ecchymoses. They may be seen in the pyramids of the kidney as well as in the hilus. Infrequently the hilus may be filled with blood. Lesions are found to be equally distributed in both kidneys. If the animal received for necropsy is dead, and had lain on one side for a period of time, the kidney (and other tissues such as the lymph nodes) on the ventral side will show increased hemorrhage as compared to the kidney or other tissues on the dorsal side. This is due to a hydrostasis of the blood, which drains from the upper tissues and engorges those beneath.

The ureters seldom show pathological changes. A few petechial hemorrhages and, on rare occasions, distention of the ureter with blood from hemmorrhages in the kidney are the only lesions observed. The urinary bladder may show a variety of hemorrhages and congestion. Mild to moderate congestion is commonly observed. A few

petechial hemorrhages are present in the majority of cases. Ecchymotic hemorrhages develop less frequently, and suffuse hemorrhages are seen only occasionally. The latter two lesions occur more commonly when secondary bacterial infection is present.

If an animal dies of hog cholera, the stomach is usually empty except for a yellow, bilious fluid and a small amount of feed or fiber. Numerous ascarids at times may be found in the stomach contents. The fundus often is markedly congested and hemorrhagic. There may be evidence of a mild to severe erosion of the mucosa. Threads of clotted blood may be attached to petechial hemorrhages in the mucosal surface.

The small intestine seldom shows more than a mild to moderate catarrhal enteritis. Mesenteric blood vessels to all intestines, however, are usually markedly engorged. Occasionally subserous ecchymotic and suffuse hemorrhages occur in either the small or the large intestines or both.

The large intestine displays a variety of lesions. Of these lesions, the most pathognomonic of hog cholera is the button ulcer. Occurring most frequently in the first part of the colon, the button ulcer is an encrusted, circular, raised lesion which appears to

FIG. 7.6—Moderate petechiae of the kidney seen most commonly in pigs infected with uncomplicated hog cholera

It is apparent that the lesions were not outstanding in more than half of the acutely and subacutely infected pigs The chronic type lesion occurred in almost 90 per cent of the cases which lingered longer than 30 days from the time of infection

Acute rib changes are sometimes easily missed Often the white line in acute cases does not show distinct enlargement, but the bone marrow just proximal to the white line may reveal a hemorrhagic band which is quite friable when touched with the point of a knife

Grossly, the subacute lesions appear to be an irregular widening of the white line at the costochondral junction At times this is not pronounced In other instances the line may be almost 2 mm in width The cartilage is separated from the bone much more easily than in normal animals The undeveloped bone at the area of separation is friable to the touch The entire area of the junction appears white

Figure 7 10 compares normal ribs, A, with hog cholera damaged ribs of pigs acutely infected, B, and subacutely in fected, C The thin white line at the costo chondral junction shown by the arrow in A is in striking contrast to the widened epiphysial lines shown by the arrows in B and C

The bone lesion in animals chronically sick with hog cholera is observed to occur as a marked transverse line of semisolid bone structure across the rib from 5-10 mm proximal from the costochondral junc tion (Fig 7 11) The animal from which these ribs were taken was ill for 38 days prior to death from hog cholera

A primary acute reaction of the disease is characterized by a marked increase in blood phosphorus and a decrease in blood cal
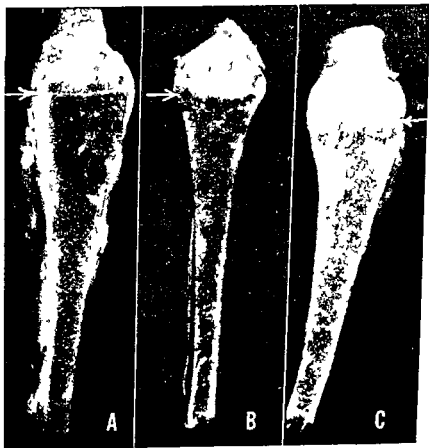

FIG 7 10—Longitudinal sec tions of ribs from (A) a normal pig, (B) a pig acutely infected with hog cholera, and (C) a pig subacutely infected with hog cholera Note the nar row regular epiphysial line (arrow) of the normal rib in A, the hemorrhagic zone in B, and wide, white line in C

sistance is lowered sufficiently The organ isms find easy access through the intestinal mucosa which is afflicted with catarrhal inflammation, petechial hemorrhages, and in farction The contents of the large intestine of the hog cholera infected pig varies from a watery yellow liquid to hard, adhering, mucus covered fecal pellets

The livers of pigs naturally infected with hog cholera are generally dark, congested, and swollen Ordinarily many ascarid scars are present giving the organ a mottled grayish appearance The gall bladder is frequently shrunken but in some cases is markedly distended The bile most often is thick and tenacious Occasionally, however, it may be quite fluid in consistency At times small, button, ulcerlike lesions have been observed on the mucosa of the gall bladder (Luedke and Dunne, 1957) Small petechial hemorrhages also may be found

Infarction of the spleen, resulting from the disruption of the flow of blood, is a lesion which is considered almost pathognomonic of hog cholera Infarctions occur as variable sized dark blebs, usually on the periphery and apex of the spleen, and are raised slightly above the surrounding surfaces (Delez, 1933)

Infarctions may occur as single lesions on the periphery or on the flat surface of the spleen (Fig 7 9) Frequently they occur as a series, coalescing to form a continuous border of infarcts along the edge of the organ At times the spleen is darkened along the periphery as if the area were infarcted but not yet fully engorged with blood In other instances, when the animal has been dead for some time before autopsy, the adsorption of hydrogen sulfide from the ad jacent intestines and the stomach causes early discoloration Congestion of the spleen occasionally may occur, but this is seen more often in other diseases such as salmonellosis and swine erysipelas The spleen becomes enlarged and darkened when congested

Frequently numerous bright red capillary tufts occur on the splenic surface, usually on the underside Some investigators have attached diagnostic significance to these bright vascular entities, but since they have been observed in apparently normal swine, their significance as a lesion of hog cholera is questioned (Kernkamp, 1939a)

Gross lesions of the brain are limited almost entirely to congestion and occasionally a few hemorrhages, primarily of the meningeal vessels At times, an increased number of bleeding points may be observed.

A disturbance of calcium and phosphorus metabolism is manifested by an interruption of bone growth at the costochondral junction in weaned pigs infected with hog cholera (Dunne et al, 1957c) The lesions are of 3 distinct types—acute, subacute, and chronic The acute and subacute lesions appear at the epiphysial line of the costochondral junction When present, the lesion is observed in most of the ribs, but is most constant in the fifth through ninth

The acute and subacute type of lesions occur most frequently In 179 acute and subacute cases, the gross lesions were observed in the following order

No gross lesions	11 7%
Mild rib changes	10 9%
Moderate rib changes	23 4%
Moderate to severe	21 7%
Severe	2 3%

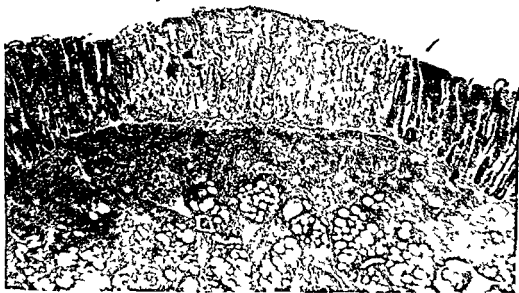FIG 7 9—Spleen showing multiple infarctions on the peripheral edges and on the flat central surface

FIG 7 12—Coagulation necrosis of a well defined area of mucosa in the colon A faint infiltration of leukocytes borders the necrosed area Hematoxylin-eosin X 60

FIG 7 13—Coagulation necrosis of the mucosa of the colon showing a well developed line of leukocytic infiltration in the submucosa and in the bordering normal mucosa Probably a later stage than in Figure 7 12 Hematoxylin eosin X 60

FIG 7 14—A late stage of necrosis of an area of mucosa in the colon Infiltrating leukocytes have forced necrotic plug of mucosa out of original position Note upward curvature of muscularis mucosa and faint outline of glandular crypts in necrotic plug Partially occluded vessels appear in submucosa Hematoxylin-eosin X 60
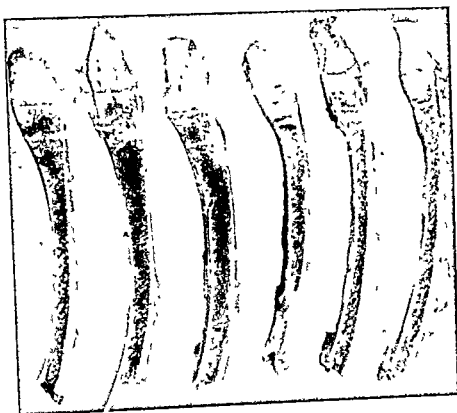
FIG. 7.11—Longitudinal sections of ribs from pigs which had been chronically ill with hog cholera, showing a transverse line of calciferous deposits approximately 1 cm. from the costochondral junction.

cium. Eveleth and Schwarte (1939) demonstrated a decrease in plasma calcium and an appreciable rise in total cell phosphorus. This was confirmed by Dunne *et al.* (1957c) who showed that the changes in concentration became evident on about the sixth day after infection.

HISTOPATHOLOGY

Primarily the virus of hog cholera attacks the reticulo endothelial system (Seifried and Cain, 1932; Bueno, 1944). In general there is a marked hydropic degeneration of the capillary endothelial cells with subsequent necrosis and hemorrhage. The sludging of blood (Beamer *et al.*, 1949) in areas of blood vessel degeneration results in the margination of leukocytes and eventual infarction. The purplish discoloration of the skin and ears is due probably to this sequence of pathological changes. According to the Seifried and Cain (1932) classification the lesions of the lymph node fall into three categories. Type 1 includes nodes in which edema and the multiplication of reticulum cells cause a separation of cellular and fibrillar elements. Follicles and germinal centers are enlarged but reduced in

atous and in some areas show perivascular infiltration with lymphocytes and histiocytes. Type 2 lesions are characterized by hemorrhages in the cell-poor substance. The peculiar distribution of the hemorrhage in the parenchyma is responsible for the marbled gross appearance. Type 3 lesions are those with advanced hemorrhage and infiltration. Erythrocytes fill the entire cell-poor substance and may cause the atrophy of lymphoid tissue.

Microscopic kidney lesions follow a pattern of hemorrhage, edema, and perivascular cuffing with macrophages and lymphocytes. Hemorrhages of smaller capillaries and glomeruli occur frequently. Tubular epithelium shows varying retrogressive changes.

The spleen offers a microscopic picture of swelling and hyalinization of blood vessel walls, obstruction with thrombotic material, and resulting infarction.

The intestinal lesion of primary interest is the button ulcer of the large intestine. Studies by Dunne *et al.* (1952b) offer evidence to support the theory that button ulcers arise from infarctions in the intestinal mucosa. The earliest stage of development is seen in Figure 7.12. A definite area of co

ation (Fig 7 16) with a few foci of microglia were found to occur with greatest frequency and severity in the thalamus and medulla (Dunne et al, 1952a) Lesions appeared to be most severe between the tenth and fourteenth day after infection

A marked histological change occurs at the epiphysis of the ribs of infected pigs The microscopic examination of the costochondral junction of a rib subacutely infected with hog cholera (Fig 7 17) shows a markedly enlarged area of mature cartilage cells (B) between the zone of cartilage cell multiplication (A) and the irregular trabecular bone (C) The irregularity of this junction of trabecular bone and the zone of lacunar enlargement is quite evident upon gross examination of the infected rib

In comparison with the rib from an infected pig a microscopic examination of the normal rib structure at the costochondral junction (Fig 7 18) reveals a small zone of lacunar enlargement (B) between the zone of proliferating cartilage (A) and normal bone marrow (C)

DIAGNOSIS

Several factors make the diagnosis of hog cholera difficult. The signs and lesions of this disease are in many ways similar to those seen in swine erysipelas, septicemic salmonellosis, pasteurellosis and streptococcosis If African swine fever were present in this country the marked similarity of its lesions to those of hog cholera would offer even greater complications to diagnosis

The inconstancy of the occurrence of lesions in hog cholera also contributes to the diagnostic dilemma Known infected pigs have been examined which showed almost none of the lesions considered diagnostic of hog cholera

The simultaneous infection of swine with hog cholera virus and other septicemic organisms offers problems of no small concern to the diagnostician The isolation of the complicating organism does not eliminate the possibility that the hog cholera virus also might be present

Making diagnosis even more difficult is the fact that the pig is the only animal known to show clinical symptoms of hog cholera The use of pigs as laboratory animals for diagnosis is limited by inconvenience and expense

No completely satisfactory laboratory test is presently available for use in hog cholera diagnosis The only tests for the presence of hog cholera virus generally accepted as diagnostic aids are the total leukocyte count to detect the presence of a leukopenia and the histological examination of the brain for perivascular cuffing Both tests have their advantages and their limitations These will be discussed later in the chapter

The culture of infecting bacteria on artificial media even when positive does not eliminate the possibility of a coexisting infection with the virus of hog cholera

FIG 7 16—Arteriole in medulla oblongata showing heavy perivascular cuffing with partial occlusion of vessel lumen Hematoxylin eosin   X 660

agulation is observed with congested anastomosing vessels conspicuous in the dying tissue. Other congested vessels are apparent in the more nearly normal tissue at either side of the dying area and in the submucosa. Leukocytic invasion of the more nearly normal tissue at the periphery of the infarcted area has just begun. There is a lack of cellular detail in the necrosed tissue; whereas, invading leukocytes and living cells are evident in the lower portion of the section. A somewhat later stage is depicted by Figure 7.13 in which an invasion of leukocytes is quite evident in the submucosa and in the living tissue at the side of the necrosing area.

A massive invasion of leukocytes in Figure 7.14 appears to have forced a plug of necrosed mucosa out of the normal position and into the intestinal lumen. Still attached, the plug of mucosa shows faint evidence of destroyed crypts, while a boundary of degenerating leukocytes and cellular debris may be observed at the base. Evidence of the eruption is further seen in the position of the muscularis mucosae which has curved upward toward the lumen in the inflammatory process. At the base of the lesion a large artery is seen. High magnification of this artery in Figure 7.15 shows

marked hydropic degeneration of endothelial cells hindering the passage of blood through the lumen.

Subsequent loss of the necrosed plug is followed by invasion of the ulcerated area by intestinal bacteria eventually resulting in the formation of the typical button ulcer.

Encephalitis is characteristic of hog cholera infection in pigs. Myelitis occurs also but to a much lesser extent. Brunschwiler (1925) described the principal microscopic lesion of the brain as a vascular and perivascular infiltration with endothelial swelling. Rohrer (1930) confirmed these findings and indicated the occurrence of these lesions in 75 per cent of the cases examined. Seifried (1931) described a mononuclear infiltration of the perivascular spaces of the parenchyma and of the spinal cord, as well as microscopical hemorrhage around the vessels not showing perivascular infiltration. Helmboldt and Jungherr (1950) found that lesions of the brain associated with hog cholera were primarily in the mesoderm and consisted of vascular and perivascular cuffs, microgliosis, leptomeningeal infiltrates, capillary hemorrhages, and hyalinization of the vascular wall. Typical perivascular cuffing and endothelial prolifer

FIG. 7.15—A vessel from base of lesion shown in Figure 7.14 demonstrating hydropic degeneration of the endothelium and partial occlusion of the lumen. Hematoxylin-eosin. X 333.

Diagnostic Signs

The signs of hog cholera were described earlier in this chapter. All signs are important in arriving at a diagnosis but some are considered particularly useful

The discharge of the eyes, which is easily detected in white pigs, and the incoordinated, 'weaving' gait are characteristic of pigs sick with hog cholera. These may occur in other diseases but to a lesser extent than in hog cholera. Infected pigs tend to pile, even in warm weather, but when standing have a depressed appearance with a drooping tail and hanging ears (most breeds). Their appetite is markedly decreased. Complete anorexia is not uncommon. Swine erysipelas infected pigs seldom pile in any weather and early in the disease have a definite bright, alert appearance. Such pigs differ from hog cholera infected pigs in that they bear their weight on their toes when standing, frequently shifting from one leg to another apparently to relieve pain. These animals soon return to a lying position. The hog cholera infected pig also differs from pigs infected with most other diseases in that it frequently vomits

Convulsions though not of common occurrence, likewise are not extremely rare. The recognition of this symptom is important not from the standpoint of a positive identification of hog cholera but rather from the fact that convulsions do not mean the positive identification of some other disease

The yellowish gray diarrhea of hog cholera is seldom seen in swine erysipelas or streptococcosis. Discoloration of the skin with hyperemia or cyanosis is common in hog cholera. Severe generalized hemorrhages of the dermis seldom occur in pure hog cholera virus infections. Primary or secondary infection with pasteurella, salmonella, or streptococci, usually are responsible for the severe dermal hemorrhages. Hog cholera infected pigs do not show a sloughing of the tail, ears, or skin except where animals previously have been exposed to *Erysipelothrix rhusiopathiae* or

some other necrotizing factor. Neither are there diamond skin lesions, welts, urticarial rash, nor swollen joints

Pigs chronically ill with hog cholera frequently display a blotching of the ears which is not constant, but when present can be considered important diagnostically. Such pigs may manifest a partial alopecia and a very scrawny appearance. Recovery is improbable

The temperatures of pigs sick with hog cholera generally range from 101°–106° F. However, both lower and higher temperatures are not uncommon. Experimentally temperatures of 109° F have been recorded but such temperatures are relatively rare. Swine erysipelas tends to cause a generally higher temperature than hog cholera early in the disease (107°–109° F.). A leukopenia is characteristic and diagnostic of hog cholera and is discussed under laboratory tests

Course of Disease

Hog cholera is not ordinarily a peracute type of disease. Deaths usually do not occur within ten days following infection whereas pigs acutely infected with swine erysipelas often die within seven days. Frequently weanling pigs infected with swine erysipelas have been found dead in the lot without having been observed to be sick. Dorset (1921) in repeated trials found that susceptible pigs simultaneously infected with filtered virus of hog cholera and live cultures of *Salmonella cholerae suis* suffered a much more acute disease than that observed following virus injection alone. Death occurred in five to seven days in the simultaneous infection

Lesions of Diagnostic Importance

Although three distinct types of lesions of lymph nodes have been described only the mottled ' lymph nodes with peripheral hemorrhage can be considered of diagnostic value in hog cholera infection. Although reported to occur infrequently in other infections, peripheral hemorrhage occurs in the mesenteric lymph nodes with marked frequency in hog cholera

Therefore, a positive bacteriological culture indicates a bacterial infection which may be either a primary infection, a simultaneous infection with hog cholera virus, or a secondary invasion of a pig already ill with hog cholera.

Case History

Hog cholera progresses through a rather definite course; and case history is quite valuable in diagnosis. Since hog cholera is the most lethal of all swine diseases it should not be overlooked in a susceptible herd. Hog cholera should be suspected if large numbers of unvaccinated pigs become ill at one time or if there is a history of one pig being sick or dying in the week preceding a general illness in the herd. In the recently vaccinated herd there may be a history of illness or death just preceding vaccination. The use of virulent virus in vaccination has at times been followed by losses which have been associated with complicating infections and heavily parasitized swine. A history of movement of swine onto the premises or the feeding of uncooked garbage from the home or from other sources may suggest hog cholera infection in a herd showing severe illness. The most convincing evidence to be obtained from case history is the report of no clinical symptoms in immune animals which are in contact with the infected herd. Also important is a history of recovering animals, for recovery is relatively rare in hog cholera.
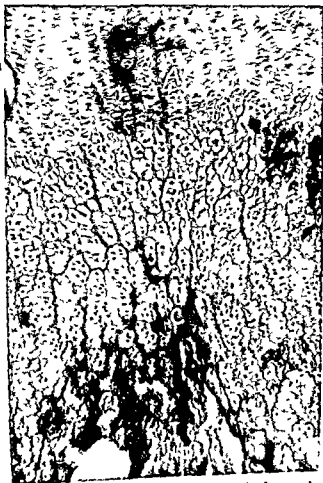
FIG. 7.17—Microscopic section of rib from pig with acute infection of hog cholera, showing (A) zone of cartilage cell multiplication, and (B) markedly increased number of cartilage cells with enlarged lacunae at costochondral junction. Note irregularity of line formed by these cells at junction of trabecular bone and zone of lacunar enlargement (C). X 55.

FIG. 7.18—Microscopic section of a normal rib at costochondral junction. Normal developing cartilage cells are shown (A). Notice regularity of epiphyseal disc and small number of cartilage cells with enlarged lacunae (B) which form the line that is observed grossly. Rapidly developing trabecular bone is evident (C). X 55.

of one and one half hours (Cole, 1932) Necrotic enteritis can cause a leukocytosis in hog cholera infected pigs (Lewis and Shope, 1929)   Primary pneumonia may delay but not prevent the occurrence of leukopenia Swine erysipelas or streptococcus infections occurring simultaneously with hog cholera tend to promote leuko cytosis (Dunne, 1948)   Swine influenza produces a mild leukopenia but not nearly to the degree seen in hog cholera (Sippel, 1952)

The microscopical examination of the brain has been shown by Helmboldt and Jungherr (1950) to be of significant value in the diagnosis of hog cholera The major limitations lie in the fact that other diseases free of active hog cholera have been shown to produce similar lesions (Jones and Doyle, 1953)   Segre (1957) used gamma globulin from the serum of hyper immunized animals and adsorbed it onto particles of the anion exchanger, Amber lite IRA-400 The particles thus coated were adsorbed onto materials containing the specific viral antigen Hog cholera was detected in the serum of experimentally infected pigs as early as 3 days after infection This test, though in an experimental stage, may prove to be useful in the diagnosis of hog cholera

Numerous other tests with varying use fulness have been reported Boynton et al (1941a) described a staining technic which disclosed inclusion bodies in the mucosal cells of the gall bladder He indicated that the bodies were demonstrable by using any polychrome stain but suggested that Kings ley s stain was most effective Sippel (1945) confirmed this work and reported that pigs in the second or third day of symptoms may not show inclusion bodies The presence of these bodies indicates hog cholera but their absence does not preclude hog cholera

In an intradermal test, Sarnowiec (1934) used castor oil and formalized blood from pigs sick with hog cholera Immune swine give an allergic response characterized by an inflammatory swelling at the area of injection Healthy susceptible pigs or those sick with hog cholera do not react to the test

A complement fixation test for hog cholera was described by Healy and Smith (1915) but their results were not confirmed by later workers

Positive proof of infection with hog cholera virus can best be obtained by the injection of a hog cholera susceptible pig and a hog cholera immune pig with blood or filtered suspension of macerated tissues from the suspected animal The hog cholera susceptible pig of course, dies and the immune pig remains well, thus giving positive identification of the disease

IMMUNITY

The immunity against hog cholera is a classical example of the three major types of resistance to infection i e , natural resistance, passive acquired immunity and active acquired immunity

Natural resistance to infection occurs on a genetic basis between species and within species All animals other than swine have a strong natural resistance to hog cholera Although this disease is one of the most lethal of all animal diseases, there usually are a few pigs in each large herd which are naturally resistant to the infection This number is small Perhaps less than 5 per cent of all swine are naturally immune to hog cholera

Passive, acquired immunity is obtained in two ways In one method the individual acquires antibodies by the injection of immune serum from another host For hog cholera this would be the serum from an other pig which was immune to the disease The other means of passive acquisition of antibodies is the passage of antibodies from the dam to the fetus or new born This is accomplished by congenital transfer through the placenta to the fetus or by maternal transfer through the mammary gland into the milk, particularly the colostrum upon which the newborn is

Hemorrhages of the kidney and urinary bladder are the most constant lesions in hog cholera but may be found in all other septicemias   Hemorrhages of the larynx once considered diagnostic are now known to occur in other diseases and are not sufficiently constant in hog cholera to be of great value

Splenic infarcts are rated as being as pathognomonic of hog cholera as any other lesion   Their usefulness in diagnosis is limited by their infrequent occurrence

Button ulcers of the large intestine have been shown to occur with relative frequency in uncomplicated hog cholera   Ulceration of the tonsils, also believed to be caused by infarction, occurs with interesting regularity in hog cholera infected pigs

The widening of the epiphyseal line at the costochondral junction of the ribs of pigs infected with hog cholera occurs with marked regularity   The absence of this lesion in all other septicemias has not been fully determined but it has not been reported to occur in swine except in pigs infected with the virus of hog cholera

Lesions which may complicate the diagnosis of hog cholera include infarcts of the kidney, necrotic enteritis suppurative pneumonia and 'paint brush' hemorrhages on the serosa of the stomach

Infarcts of the kidney are relatively rare in hog cholera. When they do occur, a complicating organism is likely to be involved   Secondary necrotic enteritis is common in hog cholera infected pigs It also occurs in salmonellosis but is usually absent in swine erysipelas or streptococcosis   Suppurative pneumonia sometimes accompanies hog cholera infection as a complicating infection but can occur as a primary disease in the absence of hog cholera It seldom is observed in swine erysipelas infection Paint brush hemorrhages are considered by some to be diagnostic of swine erysipelas, although Hofferd (1944) considered them to be of little significance These hemorrhages are seldom seen in pure hog cholera infections A markedly enlarged and dark colored spleen is usually seen in salmonellosis and may occur in swine erysipelas but is uncommon in hog cholera (Seghetti, 1946, Hofferd, 1944)

Laboratory Tests

A leukopenia associated with hog cholera was observed in early investigations of the disease (King and Wilson, 1910, Dinwiddie, 1914) Lewis and Shope (1929), Cahill (1929), and Kernkamp (1939b) emphasized the importance of the leukopenia as an aid in diagnosis

Generally, in normal pigs eight weeks of age or older, the total leukocyte count will range from 14 000 to 28,000 leukocytes per cu mm of blood Variations above and below these figures, however, have been noted in apparently normal animals, but such variations are relatively uncommon Within two to six days after exposure of a susceptible, normal pig to virulent virus the total white cell count (T W B C) will drop below 9,000 (Fig 7 2) The low point in the leukopenia may be less than 4,000 leukocytes per cu mm but commonly does not drop below 5 000 In general laboratory procedure, a leukocyte count of 9 000 or less in pigs eight weeks of age or older is considered to be positive evidence of hog cholera Since there is a tendency for a secondary leukocytosis to develop at times, following the initial leukopenia, a relatively normal or high leukocyte count does not necessarily mean that hog cholera virus is ruled out as the infecting agent This emphasizes the need for leukocyte counts on more than one animal and preferable at different stages of illness The results of this test are most satisfactory when samples are taken soon after clinical symptoms are first in evidence.

A number of factors should be considered in the interpretation of the test The normal T W B C of pigs three weeks old may be as low as 7,000 (Sippel, 1952) If an animal consumes a quantity of feed before it is bled, the T W B C may rise more than 5 000 above the normal count in a period

(1946) succeeded in altering the patho genicity of hog cholera virus by many pas sages through rabbits A resulting rabbit adapted virus proved to have marked anti genic properties in swine and showed an effective elimination of pathogenic prop erties Vaccines utilizing these adapted viruses are presently being marketed to be used with or without immune serum Commercial vaccines also are available which were developed by alternate pas sages between rabbits and swine with the final modified virus being harvested from the pig Vaccines of swine origin tend to show mild pathogenicity and are recom mended for use with immune serum Com mercially prepared vaccines are dried un der vacuum and must be reconstituted at the time of vaccination Unused portions of the mixture should not be stored for use at a later time because the virus re tains its viability for a relatively short time after being reconstituted Lapinized (rabbit adapted) live virus vaccines do not give immediate, complete protection when used without serum but at times are ca pable of effecting a resistance to infection which has the appearance of a 'virus in terference phenomenon ' This resistance has been observed as early as the second day after vaccination (Harvey and Cooper, 1954) The duration of immunity pro duced by attenuated vaccines is as good as or closely approaches, that obtained by vaccination with virulent virus and im mune serum One lapinized strain was shown to produce 100 per cent protection against death when pigs vaccinated with it were challenged with virulent virus 2 years after vaccination (Percival et al, 1953)

Killed Virus Vaccines

Effective vaccination with absolute as surance that there will be no spread of live virus is the goal of all those interested in the eradication of disease It is no won der then that the crystal violet vaccine in vestigated by McBryde and Cole (1936) should have received world wide attention The vaccine is prepared from defibrinated blood (some variations include spleen pulp and other reticulo-endothelial tis sues), to which crystal violet and glycerin are added and the mixture subjected to incubation at 37° C for a period sufficient to insure complete innocuity of the virus This vaccine was shown to protect pigs against both artificial and contact infec tion, did not transmit the disease to suscep tible animals, and produced an immunity which lasted about ten months (Doyle 1942) Crystal violet vaccine was shown to be ineffective when administered simul taneously with immune serum (Cole and Henley, 1949) D'Apice et al (1948) dem onstrated that intradermal injection of the vaccine in amounts as low as 0 5 ml pro duced a satisfactory immunity in 10 to 15 days The administration of immune serum simultaneously with crystal violet vaccine without inhibition of the antigenic proper ties of the vaccine also was reported by D'Apice and Penha (1952) Their results at this time have not been confirmed by other workers Doyle and Spears (1955) found the intradermal route to be less satisfactory than the subcutaneous route which gave better protection Torrey and Zinober (1956) reported on twelve years of vaccination with crystal violet vaccines Pigs were challenged at 122 and 188 days after vaccination and 92 8 per cent sur vived Pigs from immune sows were not as effectively immunized

A killed tissue vaccine produced from hog cholera infected swine tissues treated with glycerin and eucalyptol was reported by Boynton et al (1937 1938, 1941b) Im munity up to 6 months with a 5 ml dose was shown to be as effective as a 10 ml dose The vaccine was shown to be safe from the standpoint of spreading live virus Casselberry et al (1953) reported 98 per cent protection in tests made at 2 to 7 months after vaccination

Formalized vaccines have received con siderable attention in Europe but have not been investigated to any great extent in America

The limitations of killed virus vaccines lie in their inability to protect against hog cholera for the first two weeks after vac

fed The latter process is the one by which pigs develop resistance to hog cholera

Active immunity is acquired either by a sublethal exposure to infection or by the injection of an immunologically active form of the infectious agent Active hog cholera immunization is acquired by three main classes of biological preparations simultaneous anti hog cholera serum and virulent virus, attenuated live virus vaccines (with or without immune serum), and killed virus vaccines

Simultaneous Vaccination With Virulent Hog Cholera Virus and Immune Serum

The passive immunity obtained by the use of anti hog cholera serum (Dorset et al , 1908) provided the swine raiser with his first protection in over half a century against the disease that threatened the survival of the swine industry This serum also supplied the key for the method of simultaneous serum and virus vaccination that for fifty years was a model of vaccination procedure The active immunity produced by this system was as stable as any vaccination known

Virulent hog cholera virus for simultaneous serum and virus vaccination is obtained in the form of blood taken from pigs infected with virulent hog cholera virus The blood is usually taken on the sixth or seventh days after injection, defibrinated and preserved with phenol, the final concentration of which is one half of 1 per cent Anti hog cholera serum is prepared by injecting immunized hogs with

unpreserved whole blood virus at the rate of 5 ml of virus per pound of body weight The hog is later bled at two, three, four, and five weeks after the hyperimmunization process The blood is defibrinated and treated with an extract of navy beans and 1 per cent sodium chloride to facilitate the removal of erythrocytes (Dorset and Henley, 1916) The resulting clear serum is pasteurized 58°–59° C and then preserved with sufficient 5 per cent phenol to make the completed serum contain one half of 1 per cent phenol Active immunization is accomplished by the injection of 2–3 ml of virulent virus with hyperimmune serum in quantities ranging from a minimum of 20 ml in sucking pigs to a minimum of 75 ml in hogs weighing 180 pounds and over (Table 7 4)

As seen in Table 7 4, the amount of serum now considered necessary in the simultaneous serum virulent virus vaccination of healthy herds of swine has been markedly increased over earlier recommendations This report also suggested that the optimum time for vaccination was at four to six weeks of age (U S D A , 1951)

Attenuated Live Virus Vaccines

The attenuation of the pathogenic properties of a virus can be accomplished by a number of procedures, but to be useful as a vaccine the antigen producing properties must not be decreased Working independently, and following the procedures employed in the study of Rinderpest, Koprowski et al (1916) and Baker

TABLE 7 4

RECOMMENDATIONS FOR MINIMUM SERUM DOSES FOR USE IN SIMULTANEOUS SERUM AND VIRULENT VIRUS VACCINATION

	Reynolds (1912)	USDA—BAI (1949)	USDA—BAI (1951)
	(ml)	(ml)	(ml)
Suckling pigs	10	16	20
Pigs 20–40 lbs	15	24	30
Pigs 40–90 lbs	20	28	35
Pigs 90–120 lbs	25	36	45
Hogs 120–150 lbs	30	44	55
Hogs 150–180 lbs	35	52	65
Hogs 180 lbs and over	40–60	60	75

botulmum Type A (Graham, 1921) influenza virus (Scott, 1911), *Salmonella para typhosus* A and B (Doyle and Spray, 1920, Van Es and Olney, 1914), *Listerella mono cytogenes* (Rhoades and Sutherland, 1918) and *Pseudomonas aeruginosa* (Dunne *et al*, 1952a) likewise may be involved in a simultaneous assault upon the animal s de fense mechanism

When this occurs, the immunity pro ducing system of the animal is apparently unable to cope with the two infections at once Without adequate support from this system, the ability of the injected anti hog cholera serum to neutralize the virus is severely hampered, and the animal subse quently succumbs to the dual infection

If, for one of many reasons, the serum dosage is not adequate (as happens in the presence of variant viruses which are dis cussed further in this chapter) and second ary exposure to the pathogens named above occurs, (particularly *Salmonella cholerae suis* and *Pasteurella multocida*), many ani mals which might have survived the virus reaction die from the combined effects of the secondary invasion and the virus of hog cholera

Sometimes, the production of a satisfac tory immunity is prevented by parasitic infestations, unsanitary housing conditions, improper nutrition, or exposure to un favorable weather conditions The impor tance of parasitic infestations and poor management in vaccination failures is ac centuated by the frequency with which they are observed under these conditions

The importance of nutrition in the vac cination for hog cholera is becoming more and more apparent The susceptibility of poorly fed pigs to bacterial infections has been widely recognized by veterinarians throughout the country, but the inability of some apparently well fed swine to with stand satisfactorily the simultaneous serum and virus vaccination against hog cholera presents still another problem

Cannon (1950) is of the opinion that the depletion of body protein over a period of time, because of inadequate diet would result in the malfunctioning of the im mune mechanisms Elder and Rodabaugh (1954), however, found that feeding sows and their pigs on a ration grossly deficient in protein did not prevent immunization of the pigs against hog cholera when vac cinated with serum and virus In experi ments conducted by Reber *et al* (1951), diets which were incomplete with respect to protein were fed to pigs for a few weeks prior to infection with virulent hog cholera virus These pigs suffered a chronic course of the disease with relatively mild tissue changes Six of eight pigs recovered In the same experiment, all pigs fed adequate dietary protein died from acute hog chol era, following exposure to the virus The findings of Davies *et al* (1952) and Pond *et al* (1952) in poliomyelitis studies fur nished additional evidence that deficiencies in certain protein substances tend to de crease the adverse effects of virus infections

High levels of chlortetracycline in the feed of pigs vaccinated for hog cholera did not interfere with the immunity produced and favorably influenced the rate of weight gain There was some indication that pigs receiving the high level antibiotic in the feed had less reaction to challenge with hog cholera virus than those with lower level or no antibiotic in the feed (Smith *et al*, 1956)

To summarize the nutritional phase in vaccination, it is obvious that the relation of diet to proper immunization is not well understood It does appear that general debility as the result of dietary inade quacies may not favor the development of proper immunity in the presence of bac terial infections However swine on high protein diets are more likely to suffer severe losses if the virus injected at the time of vaccination is not adequately neutralized by the anti hog cholera serum with which it is administered While the full importance of these facts has not been clarified, it can be said that the practicing veterinarians who recommend removal of protein supplement at the time of vac cination, but who refuse to vaccinate mal

cination Thus they cannot be used where hog cholera is likely to be present, such as in garbage feeding lots and areas of en zootic hog cholera

Post-Vaccination Losses

One of the earliest recognized complications of hog cholera immunization was the occurrence of sudden deaths from shock at the time of vaccination One of the causes of this phenomenon was shown to be related to the temperatures used in the pasteurization of the serum (Munce and Hoffman, 1930) Serum heated above 60° C was shown to produce shock when injected into swine Lowering the temperature to 58° and 59° C removed this vac cination hazard

Vaccination shock also was shown to be caused by vaccination of pigs afflicted with anemia McBryde (1932) showed that there was a definite correlation between the hemoglobin content of the blood and the degree of shock following hog cholera vac cination The severity of the shock in creased as the anemia became more pronounced This was confirmed by Schipper et al (1955) who also found that no signs occurred when attenuated hog cholera vaccine or anti swine erysipelas serum was injected rather than anti hog cholera serum and virus

Many post vaccination losses, other than from shock, have been classified as virus "breaks' and serum "breaks The term virus "break" implies a failure of the virus to impart an active immunity of sufficient potency to protect against exposure to a lethal hog cholera virus after the passive immunity supplied by the serum has been depleted Since passive immunity lasts from three weeks to one month, the occurrence of hog cholera in the herd after this period suggests a failure in the production of ac tive immunity One of the primary factors contributing to this situation is the im proper handling or storing of virus Fac tors contributing to a virus 'break" in clude shipping the virus without ice, holding it too long in shipment, allowing it to stand too long at room temperature transporting it on a hot day in the trunk of a car without refrigeration or using it after it has become outdated, all of which can mean decreased viability of the virus

Excessive doses of serum also may pre vent proper active immunization Van Es and Olney (1944) demonstrated that in hog cholera immunization, serum doses of 0 43 ml per pound body weight with 2 ml of hog cholera virus permitted the best gains in pigs When 1 72 ml of serum per pound body weight were used, 2 out of 15 pigs (13 33 per cent) died of hog cholera in one experiment and 3 out of 20 pigs (15 per cent) died in another experiment upon subsequent challenge with lethal hog cholera virus The possible importance of this factor is seen, for example, when animals are sold through a community sales barn Such swine are us ually required to be vaccinated with serum or serum and virus Many times the pur chaser, to increase his safety margin, has the pigs treated again with serum and virus upon reaching the farm The double dose of serum may, in this case, neutralize the virus so completely that active im munity is not established Animals so vac cinated may be susceptible to hog cholera again as early as four weeks after vac cination To prevent this, it is suggested that a nonpathogenic, attenuated live virus vaccine, without serum, be given in place of the second injection of serum and viru lent virus

The serum ' break' is best defined as a failure of anti hog cholera serum to neu tralize the disease-producing properties of the virus with which it is administered simultaneously in vaccination Many fac tors individually may be responsible for the inadequacy of the serum Most of these have nothing to do with serum potency The most common factor is the simultane ous infection with another septicemic or ganism such as Erysipelothrix rhusio pathiae, Salmonella choleraesuis, Pasteu rella multocida or streptococci Other or ganisms, however, including Clostridium

exists in a monovalent state, and that certain specific conditions or circumstances are necessary to cause a recessive or masked form to become predominant Or, it may be simply that one strain is capable of changing so that different characteristics are recognized as antigenic or pathogenic variants The fact that some of these changes have been shown to be reversible or inconstant strengthens this latter theory However, it must be recognized that apparently irreversible changes in the pathogenicity of the virus of hog cholera have been experimentally produced in the development of the lapinized vaccines This strongly suggests that it could be possible that irreversible changes might occur naturally

Vaccination of Sows and Suckling Pigs

It is often difficult to pick a time when all animals of large swine herds are at an optimum age for vaccination Thus the following problems arise What animals can be vaccinated? Can live virus be used? When should serum alone be used? Can only part of the herd be vaccinated? Do suckling pigs develop a solid immunity?

Pregnant sow vaccination with either virulent virus or attenuated live virus should be made only in the last two months of pregnancy The vaccination of sows with live hog cholera virus in the first month after mating was found to cause ascites subcutaneous edema, and a variety of other abnormalities in the fetus prior to parturition The other abnormalities were edema of the mesocolon and perirenal tissues, mottling of the liver, asymetry of the head, lengthening and twisting of the snout and malformation of the limbs Fetal death and partial reabsorption was also noted (Sautter *et al*, 1953, Young *et al*, 1955) Killed virus preparations such as crystal violet vaccine do not produce these effects upon the fetus and can be used during the first two months of pregnancy

It is somewhat difficult to establish active immunity in baby pigs from immune sows because a certain amount of resistance is present shortly after suckling and is re

tained through the first few weeks of the pig's life McArthur (1919) found that 69 8 per cent of one day old to two week old pigs born of immune dams resisted an oral dose of 0 5 ml of virulent hog cholera virus when it was smeared on the teat of the sow Pickens *et al* (1921) injected hog cholera virus into 85 suckling pigs at ages up to and including 55 days Only 5 of 85 died These five were 40 days old when exposed to the virus and were from one litter Forty five pigs at ages ranging from 48 to 78 days were challenged with the same virus 24 to 78 hours after weaning and 41 of the 45 died

Piglets farrowed by crystal violet treated sows are susceptible to hog cholera during and after their third week of life Therefore, a good lasting immunity from crystal violet vaccine can be obtained if pigs are vaccinated at four weeks of age or older These pigs retain their immunity for nine months or longer (U S D A , 1946)

In general, if suckling pigs less than five weeks of age and from immune dams are to be immunized, it is suggested that the dose of virus be increased by 50 per cent If possible, revaccination of pigs to be kept for breeder stock should be made in 30 days

If lapinized live virus vaccines are available, they should be used without serum when vaccinating resistant suckling pigs from immune dams

TREATMENT

If observed early, it is possible to treat swine ill with hog cholera by the injection of anti hog cholera serum The amount of serum used is usually 50 to 100 per cent higher than that injected for prophylactic purposes To be effective, the serum must be given within the first 3 to 4 days after exposure to the virus If the animals have been ill for more than 4 days, serum injections are usually futile If serum is given alone, live virulent or modified virus may be given safely in ten days after the serum injections Attenuated vaccines should be given within 30 days following the serum treatment to insure continued immunity

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nourished pigs, may be vindicated by the above facts

Variant Viruses

The possibility of the existence of more than one antigenic strain of hog cholera virus poses a rhetorical as well as a scientific question which has not been satisfactorily answered

Ruppert (1930) emphasized the great variations in the virulence of hog cholera virus Hupbauer (1934), after numerous cross immunization tests, came to the conclusion that plurality of strains in hog cholera virus does not exist Nevertheless, considerable interest in the question of variant viruses was generated, following the severe post vaccination losses experienced in the 1949–50 vaccination periods The losses occurred in a widespread area throughout the midwestern United States and were believed to have been caused by a variant strain of hog cholera virus The work of Dale et al (1951) showed that commercial anti hog cholera serum, sufficiently potent to neutralize standard test viruses in swine, was incapable of neutralizing the variant strain using the same standard test doses of serum Increasing the amount of serum given from 15–45 ml was necessary to provide 100 per cent protection against the variant The virulence titer of this variant was shown to be lower than known test viruses This variant characteristic was maintained in serial passage only when anti hog cholera serum was administered simultaneously in small doses (Dale and Zinober, 1954, Dale et al, 1954) On the basis of these findings the recommendation of the U S D A Bureau of Animal Industry called for an increase of the minimum dose of serum to be used with virulent virus in vaccination for hog cholera

An editor's note (1951) stated that outbreaks of unidentified nervous disorders in swine have been reported by practitioners in widely separated locations in the corn belt' During this period a strain of hog cholera virus isolated from a pig dying in a post vaccination 'break' demonstrated

encephalitis producing characteristics which were manifested by convulsive activities in 33 of 73 infected pigs (Dunne, 1952a) This strain also showed ability to resist *in vivo* neutralization with standard test amounts of serum of known potency, but also was shown to have a somewhat higher titer than known standard test viruses The occurrence of variant virus breaks' was fairly well distributed between herds vaccinated with modified live virus vaccines tissue culture vaccines, inactivated vaccines, and virulent virus with anti hog cholera serum in tests conducted by Torrey et al (1955) In France, experiences with viruses of a variant nature were reported by Lucas et al (1953) who found that monovalent crystal violet vaccine, when given in single injections, did not protect against the variant virus but was efficacious only against the vaccinal variety The duration of immunity from the monovalent crystal violet vaccination did not seem to go beyond three months when animals were subsequently exposed to this virus A 50 to 100 per cent increase in serum dosage was necessary to neutralize the virus in vivo

'Subtypical' hog cholera was described by Kernkamp and Fenstermacher (1917) who observed infections, with a lengthy course as long as 39 days, in both vaccinated and unvaccinated herds The use of sonically vibrated virus in the development of chronic hog cholera was reported by Dunne et al (1955) The course of the disease ranged from 50 to 95 days The virus of hog cholera was isolated from these animals at death The strongest argument against the existence of plurality of antigenic strains lies in the fact that none of the 'variant' viruses described above have been shown to be capable of producing hog cholera (in significant numbers) in swine immunized for a period of 30 days or more with live virus and anti hog cholera serum or in swine surviving a natural infection

It does appear, however, that the antigenic and pathological properties of the virus of hog cholera are not as constant as was once believed It may be that no strain

Persistence of Hog Cholera Virus in Exposed Swine

Hog cholera virus has been found to be present in the blood of pigs for 14 days but not 21 days after vaccination with vir ulent virus and anti hog cholera serum The virus was found in the lymphatics in an attenuated form at 21 days but not 42 days after vaccination (McBryde, 1934) Infected "carrier" pigs were reported by Gibbs (1933) to be harboring the virus in button ulcers of the large intestine as long as 94 days after known infection with hog cholera Recovery of the virus from pigs chronically sick with hog cholera for 65 days was also reported in experiments with physically altered viruses (Dunne et al, 1955) Attempts to find 'carriers' in swine vaccinated with crystal violet vaccine and subsequently exposed to hog cholera were negative (Gwatkin and Mitchell, 1944)

Since the swine tissues that are suscep tible to hog cholera virus invasion are ap parently those of the pharynx and the respiratory tract, it seems logical that the pig s natural habit of rooting and his fond ness for garbage, particularly animal pro tein, should provide an optimum oppor tunity for infection Uncooked garbage, either of commercial origin or from the farmhouse kitchen, offers the prime sources of infection Severe endemics in areas pre viously free of the disease have been ex perienced in Canada as the result of feed ing uncooked swill (Hall, 1952) Birch (1917) demonstrated that the virus of hog cholera could survive the curing process of hams and was not destroyed in refrigerated carcasses Bacon prepared from infected pigs was capable of causing hog cholera after 27 days but not after 37 days (Edgar et al, 1952) The virus can survive for at least 73 days in the bone marrow of salted pork (Doyle 1933) Perhaps one of the most simple yet most effective methods of preserving the virus is by freezing Infected pork was capable of causing hog cholera after 1 598 days of refrigeration at −11° C (Edgar et al, 1952)

The influence of the hydrogen ion con centration on the survival of hog cholera virus was studied by Chapin et al (1939) who showed that it survived best at a pH of 4 8–5 1 Although the virus is relatively stable to short periods of ultraviolet ir radiation (Bell 1954), it is believed that sunlight has an active part in its inactiva tion under natural conditions Heat also is an important virus inactivator Ray and Whipple (1939) found that phenolized virus maintained without refrigeration for any appreciable time in the trunk of an automobile under the direct rays of the sun was readily rendered avirulent Virus with an initial titer of 1×10⁻⁸ dehydrated by freeze drying and stored at 4° C in sealed waterproof bags was still viable at dilutions of 1×10⁻⁴ after more than two years (Schwarte and Mathews 1954a) Blood frozen in screw cap vials at −10° C was virulent after more than 6 years of storage (Dunne et al, 1957b)

In comparison to other viruses, the virus of hog cholera is relatively stable to chemical disinfectants Kresco ' a coal tar disinfectant was effective in 5 minutes in 2 per cent concentration but not in one per cent concentration (King and Drake 1916) Sodium hydroxide in 3 per cent concentration with 2 per cent lime water was effective in 15 minutes (McBryde et al, 1931) A pH of 1 4 or 13 0 is neces sary to kill the virus within one hour (Slavin 1938) Phenol in 5 per cent solu tion and hypochlorite solution containing 1 66 per cent available chlorine each de stroyed the virus in 15 minutes Less con centrated solutions of these chemicals re quire longer periods than are indicated above to acquire the desired results

Control and Eradication

The United States has attempted the control of hog cholera for a period of al most 50 years The simultaneous virulent virus and immune serum method of vac cination has prevented the destruction of the swine industry by effecting a partial control of the losses due to hog cholera In this method of control, however, the propa gation of the disease by vaccination breaks has been a factor in maintaining

EPIZOOTIOLOGY AND CONTROL

The epizootiology of hog cholera infections is not completely understood The virus appears to be easily transmitted from pig to pig but its sudden appearance in remote places, often where there is no history of hog cholera, poses a problem for which there is no complete answer

The direct contact of a susceptible pig with an infected pig offers one of the most positive methods for the introduction of hog cholera into a herd The introduction of newly purchased swine into the herd, without an adequate isolation period is a common error in management Transportation of animals in trucks or other vehicles that have not been properly cleaned and disinfected contribute to the spread of hog cholera Public auctions may be a clearing house for infected animals Such animals are not always easily detected Vaccination with virulent virus offers a possible source of infection, if the conditions for vaccination are not sufficiently good Although this system of vaccination has been responsible for the control of hog cholera for fifty years its part in the continued prevalence of the disease is also recognized (Atherton, 1923)

Epizootiologists in general are of the opinion that a natural reservoir of infection exists in nature which is responsible for the pockets of infection which seem to come from nowhere Shope (1957) is of the opinion that a close similarity may exist between hog cholera and swine influenza with the earthworm playing an important role in both infections Transmission of hog cholera by house flies was shown to be possible by Dorset *et al* (1919), who permitted flies to feed on eye excretions from infected pigs At intervals up to and including 24 hours after removal from the excretions, the flies were ground and injected into susceptible swine In every case transmission was shown to be possible

Apparently, close proximity between infected and susceptible animals is not enough to insure transmission of hog cholera under experimental conditions In three adjacent outdoor pens, Porter (1923) conducted transmission experiments to show the ability of chickens to mechanically transmit the disease Solid fences three feet high separated the three pens Pen One contained susceptible swine only Pens Two and Three each had three pigs and were enclosed with a fine mesh wire Six leghorn chickens were permitted to fly between Pens Two and Three Infection initiated in Pen Two was transmitted to Pen Three but Pen One remained uninfected at 20 days Dorset (1916) observed that if pens were placed 50 feet apart, transmission did not occur when caretakers walked through a pen with infected pigs and then walked into a pen with susceptible pigs In another situation pigs confined in a building under what was considered excellent isolation conditions experienced two 'breaks in spite of all the precautions taken (Hoskins 1917)

The length of time that a pen which has contained infected pigs will remain infectious for susceptible pigs has not been completely investigated Dorset (1916) and Edgar *et al* (1952) revealed that the virus of hog cholera was not very stable under the conditions which prevailed when the experiments were made Both groups were unable to demonstrate the presence of virus in pens which were allowed to stand 48 hours after the removal of sick animals. The Australian workers even plugged the drains to retain all infectious material The virus was detected in 24 hours but not in 48 hours Whiting (1926) found that virus in outdoor pens remained viable for at least two days in November but only one day in the summer Transmission between pens was prevented by placing cheesecloth above the partitions

Survival of the virus in dung under various conditions has varied from 2 to 4 days depending upon the heat generated. Virus could not be detected in manure water collected from pens containing infected pigs In experimentally contaminated manure water, the virus survived from 2 to 7 weeks The hydrogen ion concentration was not determined (Geiger 1933)

CANNON, P. R. 1950 The role of proteins in relation to resistance to infection Jour Amer Vet Med Assn 116 451

CASSELBERRY, N. H., MALMQUIST, W. A. HOLLIHAN, R. B. AND BOYNTON, W. H. 1953 Hog cholera immunization with a new vaccine propagated in vitro and hog cholera antiserum Vet. Med. 48 24

CHAPIN, R. M POWICK, W. C., McBRYDE, C. N., AND COLE, C. G 1939 The influence of hydrogen ion concentration on the survival of hog cholera virus in defibrinated blood Jour Amer Vet. Med Assn 95 494

COLE, C. G 1932 Leucocyte counts on the blood of normal cholera infected and recently immunized pigs Jour Amer Vet. Med Assn 81 392

——, AND HENLEY, R. R 1949 Experiments on the combined use of crystal violet vaccine and the anti hog-cholera serum in the prevention of hog cholera. U.S.D.A Circ. No 807, pp 1 12

——, AND HUBBARD E D 1946 Concentration of hog-cholera virus in the blood of artificially infected swine at different stages of the disease Jour Amer Vet. Med Assn 108 143

DALE, C. N., SCHOENING, H W COLE C G HENLEY R R AND ZINOBER, M. R. 1951 Variations (variants) of hog cholera virus. Jour Amer Vet. Med Assn 118 279

——, AND ZINOBER, M R. 1954 Variations (variants) of hog cholera virus II Perpetuation and attempts at enhancement of variant characteristics of hog cholera virus by means of serial passage with antiserum and without antiserum Jour Amer Vet. Med Assn 125 137

——, AND TORREY, J P 1954 Variations (variants) of hog cholera virus. III Further attempts to enhance its variant characteristics by simultaneous passage with varied amounts of different serums Proc. 91st Ann. Meet. Amer Vet Med Assn, p 124

D'APICE, M., AND PENHA, A H 1952 Experiencias de sero vacunacion simultanea con vacune de cristal violeta por via intradermica Rev Vet Med. 34 1

——, AND CURY, R. 1948 Vaccination against hog cholera with crystal violet vaccine by the intradermic route. Amer Vet Med Assn 112 230

DAVIES W L., POND, W L, SMITH S C., RASMUSSEN A F JR., ELVEHJEM, C. A., AND CLARK, P F., 1952. The effect of certain amino acid deficiencies on Lansing poliomyelitis in mice Jour. Bact. 64 571

DELEZ, A. L 1933 Splenic lesions in hog cholera Jour Amer Vet Med Assn 83 82.

DE SCHWEINITZ, E. A., AND DORSET, M 1903 New facts concerning the etiology of hog cholera. U.S.D.A., Bur Anim Ind. 20th Ann Rep., p 157

DIMOCK, W W 1916 Symptoms, lesions and differential diagnosis. Jour Amer Vet Med Assn 48 213

DINWIDDIE, R R 1914 Studies on the hematology of normal and cholera infected hogs Univ Ark. Agr Exp Sta Tech Bull 120

DORSET, M 1916 Review of research work on hog cholera Proc. 20th Ann Meet U S Live stock San Assn p 42

—— 1921 Report of experiments with suipestifer bacterins. Proc. 25th Ann. Meet. U S Livestock San Assn p 146

—— 1922. A note on the period of incubation of hog cholera Jour Amer Vet. Med Assn 61 393

—— BOLTON B M., McBRYDE, C. N 1904 The etiology of hog cholera U.S.D.A., Bur Anim Ind. 21st Ann. Rep., p 138

—— AND HENLEY, R. R. 1916 Production of clear and sterilized anti hog-cholera serum Jour Agr Res. 6 333

—— McBRYDE, C. N., AND NILES W B., 1908 Further experiments concerning the production of immunity from hog cholera U.S.D.A., Bur Anim Ind Bull. 102.

—— , AND RIETZ, H., 1919 Observations concerning the dissemination of hog cholera by insects. Vet Med 12 55

DOYLE, L. P., AND SPRAY, R. S., 1920 Pathogenic bacteria in hog cholera blood. Jour Infect Dis. 27 215

DOYLE, T M 1933 The viability of the virus of swine fever in bone marrow muscle and skin of preserved carcasses. Jour Comp Path. and Therap 46 25

—— 1942 Crystal violet vaccine for the prevention of swine fever Vet Jour 98 51

—— AND SPEARS H N 1952 Injection of crystal violet swine fever vaccine into the ear Vet Rec. 67 108.

DUNNE, H. W. 1918 White blood cell determinations in hog cholera cases complicated with pneumonia Mich St Coll Vet S 127

—— BENBROOK S. C., SMITH E. M. AND RUNNELLS, R. A. 1957c Bone changes in pigs infected with hog cholera Jour Amer Vet. Med. Ann. 130 210

—— AND LUEDKE, A J. 1957 Unpublished data

—— , AND HOKANSON J F 1957c Unpublished data

—— , AND REICH C. V. 1957a The in vitro growth of hog cholera virus in cultures of peripheral blood Amer Jour Vet Res 18 502.

—— REICH C. V., HOKANSON J F., AND LINATOUR E. S 1955 Variations of hog cholera serum A study of chronic cases. Proc. Book Amer Vet Med Assn 92nd Ann Meet. p 149

hog cholera as an endemic disease within the nation No program for the eradication of hog cholera can be effective where virulent virus is used in the vaccination procedure It is even questioned whether adequate control is possible under the same conditions The elimination of virulent virus from use in hog cholera vaccination would be a necessary major step in the eradication of the disease The popularity of attenuated and killed virus vaccines is indicated by recent figures from the Committee on Nationwide Eradication of Hog Cholera (Milligan, 1956) which show that 72 per cent of all swine vaccinated in the United States in 1955 were immunized with attenuated or killed vaccines while only 28 per cent were vaccinated with virulent virus and immune serum There also is evidence that a decline in the incidence of hog cholera may have occurred since 1953 The cases of hog cholera at the Iowa Veterinary Medical Diagnostic Laboratory are reported as follows 1953, 330 cases, 1954, 200 cases, and 1955, 132 cases (Schwarte, 1956)

A major factor in the decline of hog cholera in this country has been the cooking of garbage More than 90 per cent of the commercial garbage fed to pigs is being cooked before feeding Swine raisers must be educated, however, that pork scraps from the farm kitchen also must be cooked

The need for effective quarantine of hog cholera infected animals is imminent. Effective measures are necessary to prevent transportation of infected animals to slaughter, to community sales, or to other points of public dispersion It has been common practice to reduce financial losses by shipping exposed hogs quickly to market. Such animals provide infected meat scraps for wide distribution Movement of swine in areas where the disease is present must be prohibited to effect eradication of hog cholera

Thorough disinfection of trucks and railway cars used for animal transportation as well as hog cholera exposed pens and houses, is necessary to stamp out the disease

Any program for eradication would be ineffective without an efficient educational program for the swine raiser The information of why, how, and when is essential to obtain the desired goal of a hog cholera free nation

REFERENCES

ATHERTON J K 1925 *Hog cholera control versus prevention* Jour Amer Vet Med Assn 17 278

BAKER J A 1946 Serial passage of hog cholera virus in rabbits Proc Soc. Exper Biol and Med 63 183

BEAMER P D BLOCK E H WARNER L BROOKS F ANLIKER J A, AND KNIPLL M H 1949 Sludged blood in three young pigs experimentally infected with hog cholera Amer Jour Vet Res 10 111

BELL, W B 1951 Studies of the Hog Cholera Virus I The effect of ultraviolet irradiation Vet Med 49, 17 18

BIRCH R R 1917 Hog cholera transmission through infected pork Jour Amer Vet Med Assn 1 303

BOYNTON, W H 1946 Preliminary report on the propagation of hog cholera virus in vitro Vet Med H 316

———— Woods G M, AND Wood F W 1937 Progress in hog cholera control with tissue vaccine Jour Amer Vet Med Assn 90 51

————, AND ———— 1938 Field application of hog cholera tissue vaccine Jour Amer Vet Med. Assn 93 291

———— AND CASSELBERRY, N H 1941a Cell changes in the gall bladder as an aid in the diagnosis of hog cholera Proc 45th Ann Meet U S Livestock San Assn p 11

————, AND ———— 1941b Immunological studies with hog cholera tissue vaccine Proc 45th Ann Meet U S Livestock San Assn p 1

BRUNSCHWILER A 1925 Ueber Meningitis acuta and serwan the Zustände beim Schwein Ie. nht Infektionskrankh. parasit Krankh u Hyg d. Haustiere und 277

BUENO P 1941 Estudos sobre a peste suina I O papel da associação em criobania Arq Inst Biol., São Paulo 15 81

———— 1944 Estudos sobre a peste suina II A reação do sistema reticulo endotelial Arq Inst Biol São Paulo 15 87

CAHILL, E A 1929 Post vaccination trouble A possible diagnostic method Jour Amer Vet Med Assn 74 123

McArthur C L 1919 Transmissibility of immunity from mother to offspring in hog cholera Jour Infect Dis 24 45

McBryde C N 1932 Anemia in relation to vaccination shock in young pigs Jour Amer Vet Med Assn 81 582

—— 1934 The persistence of hog cholera virus in the bodies of swine after simultaneous inoculation Jour Amer Vet Med Assn 84 420

—— AND Cole C G 1936 Crystal violet vaccine for the prevention of hog cholera A progress report Jour Amer Vet Med Assn 89 652

—— AND Niles W B 1929 A study of the simultaneous and serum alone methods in the treatment of cholera infected hogs Jour Amer Vet Med Assn 74 153

—— —— AND Cole C G 1931 Experiments to determine the effect of sod ium hydroxide and calcium hydroxide on the virus of hog cholera Jour Amer Vet Med Assn 79 87

Maitland M C AND Maitland H B 1931 Cultivation of foot and mouth disease virus Jour Comp Path and Therap 44 106

Maurer F D 1956 Personal communication

Milligan J 1956 Report of the committee on nationwide eradication of hog cholera Proc 60th Ann Meet U S Livestock San Assn p 270

Munce T W AND Hoffman H A 1930 Vaccination shock in young pigs (anti cholera serum shock) No Amer Vet 11 37

Percival R C Harvey M J James T AND Koprowski H 1953 Studies on modified hog cholera vaccine Duration of immunity Vet Med 48 359

Pickens E M Welsh M F AND Poelma L J 1921 The susceptibility of young pigs to hog cholera Jour Amer Vet Med Assn 58 403

Pond W L Davies W L Smith S C Elvehjem C A Rasmussen A F Jr AND Clark P F 1952 The influence of amino acid deficiencies on Theiler s GD VII Encephalomyelitis of mice Jour Bact 64 583

Porter E W 1923 Some experimental work in hog cholera Jour Amer Vet Med Assn 63 580

Quin A H 1950 The past and future of hog cholera control Jour Amer Vet Med Assn 116 411

Ray J D AND Whipple G E 1939 Effects of heat on phenolized hog cholera virus Jour Amer Vet Med Assn 95 278

Reagan R L Brueckner A L AND Poelma L J 1951 Morphologic studies of hog cholera virus by electron microscopy Amer Jour Vet Res 12 116

Reber E Whitehair C K AND MacVicar R 1951 Nitrogen metabolism of hogs infected with hog cholera virus Fed Proc 10 239

Reynolds M H 1912 Hog cholera serum work with special reference to disappointments Proc Amer Vet Med Assn p 519

Rhoades H E AND Sutherland A K 1918 Concurrent Listerella monocytogenes and hog cholera infections Jour Amer Vet Med Assn 112 151

Rohrer H 1930 Histologische Untersuchungen bei Schweinepest Veranderungen im Zentralnervensystem in akuten Fallen Arch wiss prakt Tierheilk 62 139

Ruppert F 1930 Über Virulenzschwankungen des Virus der Schweinepest Messner Festschrift Fritz Eberlein Hanover

Salmon D E 1889 Hog cholera its history nature and treatment as determined by the inquiries and investigations of the Bureau of Animal Industry U S Govt Print Office Washington D C

Sarnowiec W 1934 Au sujet de la nouvelle méthode de diagnostic de la peste du porc Bull Acad vét France 7 201

Sautter J H Young G A Luedke A J AND Kitchell R L 1953 The experimental production of malformations and other abnormalities in fetal pigs by means of attenuated hog cholera virus Proc 90th Ann Meet Amer Vet Med Assn p 146

Schipper I A Bolin F M AND Eveleth D F 1955 Vaccination induced shock of anemic pigs Vet Med 50 61

Schwartz L H 1955 The migration of hog cholera virus when subjected to electrophoresis Jour Amer Vet Med Assn 40 177

—— 1956 Incidence of hog cholera in Iowa during the past year and studies made on current field problems Vet Med 51 539

——, AND Matthews J 1953a Stability of hog cholera virus Vet Med 49 375

—— AND —— 1953b Transmission of hog cholera via the respiratory tract No Amer Vet 35 671

Scott J P 1911 Swine influenza associated with hog cholera Vet Ext Quart April 1911

Seghetti L 1916 Observations regarding Salmonella choleraesuis (Var kunzen dorf) septicemia Jour Amer Vet Med Assn 109 134

Segre D 1957 A new serological test for the detection of viral antigens Abst Fed Proc 16 132

Seifried O 1931 Histological studies on hog cholera I Lesions in the central nervous system Jour Exper Med 53 277

——, AND Cain C B 1932 Histological studies on hog cholera III Lesions in the various organs Jour Exper Med 56 331

DUNNE H W   SMITH E M   AND RUNNELLS R A   1952b   The relation of infarction to the formation of button ulcers in hog cholera infected pigs   Proc. Book Amer Vet Med Assn 89th Ann Meet   p 155

———   STAFSETH H J   AND THORP F JR.   1952a   A study of an encephalitic strain of hog cholera virus   Amer Jour Vet Res   13 277

EDGAR G   HART L   AND HAYSTON J T   1952   Studies on the viability of the virus of swine fever   Rep 14th Int Vet. Cong 2 387

EDITOR'S NOTE   1951   Vet Med   46 177

ELDER C   AND RODABAUGH D E   1954   Role of protein in immunization of swine against cholera   Univ Mo Agr Exp Sta Res Bull 621

EVELETH D F   AND SCHWARTE L H   1939   Chemical changes in the blood of swine infected with hog cholera   Jour Amer Vet Med Assn   94 411

FRENKEL S   VAN BEKKUM J G   AND FRENKEL H S   1955   La culture du virus de la peste porcine dans le tissu splenique du porc explanté en milieu liquide   Bull Off Int Epiz 43 323

GEIGER W   1933   Die Haltbarkeit des Virus der Schweinepest in Dünger und Jauche   Deutsch tierärztl Wschr   41 625

GIBBS C S   1933   Filtrable virus carriers   Jour Infect Dis   53 169

GOLDMAN G   AND PEHL K H   1955   Über die Vermehrung des Schweinepestvirus in der Sauglingsmaus   Arch Exper Vet Med   9 732

GRAHAM R   1921   Botulism in swine and its relation to immunization against hog cholera   Jour Amer Vet Med Assn   60 76

GUSTAFSON D P   AND POMERAT C M   1956   Cytopathic changes in tissue cultures derived from a pig infected with hog cholera   Amer Jour Vet Res   17 165

GWATKIN R   AND MITCHELL C   1944   Studies on swine fever II   Search for carriers in vaccinated and exposed animals   Canada Jour Comp Med   8 350

HALL O   1952   Garbage feeding control in Canada   Proc. 56th Ann Meet U S Livestock San Assn   p 209

HANSON R P   1956   The origin of hog cholera   Jour Amer Vet Med Assn 131 211

HARVEY M J   AND COOPER F   1954   Effect of exposure to hog cholera virus before and after vaccination with modified live virus vaccine   Jour Amer Vet Med Assn   124 111

HEALY D J   AND SMITH W V   1915   Compliment fixation in hog cholera   Jour Infect Ds 17 213

HECKE F   1932   Die künstliche Vermehrung des Schweinepestvirus mittels Gewebekulturen   Zentralbl f Bakt I Abt Orig 126 517

HELMBOLDT G F   AND JUNGHERR L L   1950   The neuropathologic diagnosis of hog cholera   Amer Jour Vet Res   11 11

HOFFERD R M   1941   Diagnosis and control of some communicable swine diseases   Cornell Vet   31 152

HOSKINS H P   1916   Notes on the occurrence of petechial hemorrhages in the larynx and kidneys in hog cholera   Jour Amer Vet Med Assn   49 178

———   1917   Hog cholera transmission experiments   Circ Res Lab   Park Davis and Co

HUBAUER A   1931   Zur Frage der Pluralität des Schweinepestvirus   Zeitschr f Infektionskrankh parasit Krankh u Hyg d Haustiere   45 291

———, AND SKOKOVIC, L.   1938   Prilog epizootiologiji suinjske kuge   Da li ouce igraju kod šrenja suinjske kuge trajesnu ulogu?   Vet. Arhiv 8 453   Abst Vet Bul 9 169   1939

JACOTOT H   1937   Sur le domaine zoologique du virus de la peste porcine   Bull Acad Vet France 10 80

———   1939   Sur la transmissibilité de la peste porcine à diverses espèces animales   Ann Inst Pasteur 62 516

JONES R A   AND DOYLE L P   1953   A study of encephalitis in swine in relation to hog cholera   Amer Jour Vet Res   52 415

KERNKAMP H C H   1939a   Lesions of hog cholera   Their frequency of occurrence   Jour Amer Vet Med Assn   95 159

———   1939b   The blood picture in hog cholera   Jour Amer Vet Med Assn   95 525

———   AND FENSTERMACHER R   1947   Subtypical Hog cholera   Proc 51st Ann Meet U S Live stock San Assn   p 96.

KING W E.   AND DRAKE R. H   1916   The action of a coal tar disinfectant on hog cholera virus.   Jour Amer Vet Med Assn   18 315

———   AND WILSON R H   1910   Studies in hog cholera preventive treatment II   Hematological stud es   Kansas State Agr Exp Sta Bull   171 159

KOBE K   AND SCHÜTT W   1931   Differential diagnose zwischen chronischer Schweinepest und Ferkelgrippe   Deutsch tierärztl Wschr 42 145

KOJROWSKI H   JAMES T R   AND COX H R   1946   Propagation of hog cholera virus in rabbits.   Proc. Soc. Exper Biol and Med 63 178

LEWIS P A   AND SHOPE R E.   1929   The blood in hog cholera   Jour Exper Med   50 719

LUCAS A.   BOULEY G   LARAF A.   AND QUINCHON C.   1953   Variation du virus de la peste porcine en France   129 18

LUSBAR A J   AND DUNNE H W   1957   Unpublished data

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African Swine Fever
(East African Swine Fever, Wart Hog Disease)

African swine fever (pestis africana suum, maladie de Montgomery, varkpes) is an acute, febrile, highly contagious viral disease of swine It is characterized by a short course, a very high mortality, and gross lesions that closely resemble those of acute hog cholera This virus produces an inapparent transmissible infection in wart hogs and other wild swine which serve as carriers No other species has been found to be susceptible The causative virus of the disease is immunologically distinct from that of hog cholera, and animals immune to hog cholera are fully susceptible to African swine fever

HISTORY

Montgomery (1921) reported that East African swine fever was seen first in East Africa about 1910, and its differentiation from hog cholera was revealed by the susceptibility to African swine fever of animals that were hyperimmune to hog cholera Steyn (1928), in his description of swine fever in South Africa, pointed out its similarity to East African swine fever

From these early reports it appears that by 1926 East African swine fever was present in both East and South Africa, the disease still is prevalent in both of these areas The severity of some of the early outbreaks in South Africa is indicated by DeKock et al (1940) They described an outbreak involving 11,000 animals in 1933 and 1934 Of these, more than 8,000 died about 2,000 were slaughtered in an emergency program designed to control the disease, and only 862 were considered survivors

DISTRIBUTION

In addition to East and South Africa, African swine fever occurs in West Equatorial Africa, where it precludes the commercial raising of swine in some regions Fortunately the disease has not been reported outside of the African continent.

ETIOLOGY

African swine fever is caused by a filtrable virus which can be demonstrated in the blood, tissue fluids internal organs and all excretions and secretions of infected animals Whole blood or splenic tissue harvested during the third or fourth day that the animals temperature is higher

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SHOPE, R. E · 1957. Personal communication.

SIPPEL, W. L : 1945. The Boynton gall bladder smear for diagnosing hog cholera. Cornell Vet. 35·147.

———: 1952. White blood cell count in hog cholera. Vet. Med. 47:497.

SLAVIN, G : 1938. The resistance of the swine fever virus to physical agencies and chemical disinfectants. Jour. Comp. Path. and Therap 51·213.

SMITH, H. R , FERGUSON, L. D., AND SANGER, V. L.: 1956. The effect of high levels of aureomycin (chlortetracycline) in the ration on development of immunity following vaccination against hog cholera. Jour. Amer. Vet. Med. Assn 129.162.

TORREY, J. P., AND ZINOBER, M. R : 1956. Twelve years successful vaccination of farm herds with crystal-violet glycerol hog cholera vaccine. Proc. 60th Ann. Meet. U. S. Livestock San Assn. In press.

———, ———, AMTOWER, W. C. AND GITZ, G. H.: 1955. Studies on modified virus vaccines for hog cholera. I. Losses in farm herds after vaccination. Proc. 59th Ann. Meet. U. S Livestock San. Assn, p 343.

U.S D.A.: 1887–88. Bur. Anim. Ind. 4th and 5th Ann. Rep , p. 274.

——— 1946. Report of Chief of Bur. Anim. Ind. Supt. of Documents U S. Govt. Print. Office Washington, D C.

———. 1949. Bur. Anim. Ind. Code of Federal Regulations, p. 118 8.

———: 1951. Bur. Anim. Ind. Code of Federal Regulations, CFR Amendment, p. 119 63

VAN ES, L., AND OLNEY, J. F.: 1944. Collected observations pertaining to hog cholera. Univ. Nebr. Agr. Exp Sta. Res. Bull. 135.

VECHIU, AL: 1939 Sur la transmission de la peste porcine à d'autres espèces animales. Bull. Off. Int. Epiz. 18:167.

WHITING, R A: 1926 Hog cholera studies Jour. Infect Dis. 38.256

YOUNG, G. A , KITCHELL, R. L, LUEDKE, A J, AND SAUTTER, J. H., 1955 The effect of viral and other infections of the dam on fetal development in swine. I. Modified live hog cholera viruses — Immunological, virological and gross pathological studies Jour. Amer. Vet. Med. Assn. 126 165.

ZICHIS, J : 1939. Studies on hog cholera virus. Jour. Amer. Vet. Med. Assn. 95.272.

taminated food and water Numerous field outbreaks in South Africa have been attributed to the practice of feeding uncooked garbage containing infective pork trimmings to domestic swine

Under field conditions the African swine fever virus is carried by wart hogs (Phacochoerus) and bush pigs (Potamochoerus) in which it produces an inapparent infection Contact between these infected wild swine and domestic species has initiated many of the outbreaks in Africa Once established in domestic swine, the infection spreads rapidly by direct contact or through contaminated feed Some field experience suggests that an insect vector may be involved in transmission, but experimental evidence is lacking Walker (1933) removed swine lice and fleas from infected animals and placed them on susceptible swine, but the disease was not transmitted The injection of virulent material subcutaneously, intramuscularly, intraperitoneally, intravenously, or intranasally has invariably produced the disease in susceptible animals

CLINICAL CHARACTER

The incubation period following contact exposure is 5 to 9 days the shorter period being the more common Experimental infection is clinically apparent in 2 to 5 days

The onset of the disease is marked by an abrupt rise in temperature to more than 105° F where it remains for approximately 4 days Distinct clinical signs usually are not apparent until the temperature begins to decline about 48 hours preceding death (Fig 8 1) This characteristic delay in the development of the clinical features until the temperature curve starts downward is in contrast to the situation in hog cholera in which clinical signs appear as the temperature rises During the first 3 or 4 days of fever, the animals usually appear bright and continue to eat and move about normally Within the 48 to 36 hours preceding death they stop eating, become obviously depressed, and usually lie huddled together in a corner When forced to move about, they do so reluctantly and exhibit profound weakness especially in the hind legs The pulse is extremely rapid Cough and accelerated respiration or dyspnea appear in about one third of the cases Serous to mucopurulent conjunctival and nasal discharges may be present With some strains of virus diarrhea, which is occasionally bloody and vomiting

Clinical Chart — African Swine Fever
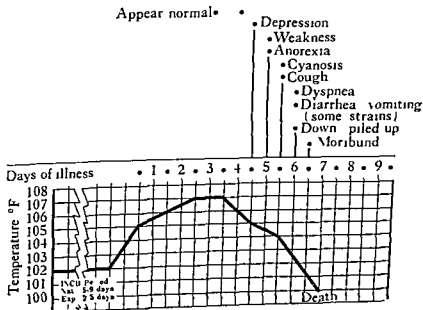
FIG 8 1—Diagrammatic representation of the sequence of symptoms in African swine fever

than 104° F will usually contain from 10⁶ to 10⁷ infective doses per gram when administered intramuscularly to pigs The virus is exceptionally stable DeKock *et al* reported survival of the virus in blood stored in a cold dark room for 6 years The virus in blood will survive for several weeks at room temperature, and contaminated pens in the tropics should be considered infectious for at least two weeks In Walker's (1933) experience, the virus survived in a filtrate of decomposed blood 16 days old and in unfiltered decomposed serum 106 days old Heating infective blood at a temperature of 55°C for 30 minutes or of 60°C for 10 minutes destroyed the virus The addition of formalin in a 1 100 concentration killed the virus in blood within 6 days A 15 per cent solution of trypan blue neither killed nor attenuated the virus within 48 hours Undiluted Lugol's solution killed the virus in blood in 10 minutes

The virus has been propagated by McIntosh (1952) in the yolk sac of 8 day embryonated eggs He started egg passages with virus which had been through a series of alternating pig and rabbit passages, and when he reported the work in 1952, the virus had been carried for 12 egg passages Subsequently it was reported by Henning (1956) that propagation of the virus in chick embryos was being continued, but that serial passage in embryos could be maintained only by an occasional passage through the pig A few pigs had survived infection with the egg propagated strain and were found to be immune to the homologous strain but not to a strain obtained from the Belgian Congo

Inasmuch as there has been a dearth of survivors, immune serum seldom has been available Therefore, very little well established information has been obtained on the immunity produced by the African swine fever virus or on the antigenic characteristics of different strains Attempts by Walker (1933) to produce a vaccine, although unsuccessful, suggested that the virus was capable of changing its antigenic character after a few animal passages Walker's experience also indicated that recovered animals are immune only against the homologous virus that produced the infection Clinically recovered animals have carried the infective virus for at least 10 months When convalescent sera from such recovered animals are heated and injected in large volume, with a minimal infective dose of African swine fever virus, test swine seldom survive Such convalescent sera have demonstrated a very low protective titer even against the homologous virus Virus strains vary in virulence, and the information available indicates that strains also differ antigenically No comprehensive immunological comparison of different strains has been reported, nor have studies been made to determine their antigenic stability No serologic test is available for diagnosis, and a vaccine has not yet been developed.

HOST RANGE

Domestic and wild swine are the only animals known to be naturally susceptible to African swine fever Domestic swine of all breeds and ages appear to be fully susceptible Attempts to infect white mice, guinea pigs, rabbits, cats, dogs goats, sheep, cattle, horses, and doves by Montgomery (1921), Steyn (1928), and Walker (1933) were reported to be unsuccessful McIntosh (1952), however, referred to the work of Neitz and Alexander, who made a limited number of serial blind passages in rabbits Velho (1956) reported that the virus produced lethal infection in swine after 26 serial blind passages in rabbits.

TRANSMISSION

The persistence of the virus in high titers in the tissues and fluids of infected and convalescent swine, the exceptionally high resistance of the virus to putrefaction high temperatures, and drying, and the susceptibility of all domestic swine are factors that facilitate transmission of the disease Natural infection is produced most readily by direct contact with infected animals, but may also result from exposure to infected premises and ingestion of con-

inguinal regions. Interlobular and subpleural edema is often prominent and epicardial hemorrhages are striking.

Lymph nodes. In African swine fever the lymph nodes provide some of the most distinctive lesions. The visceral nodes are hemorrhagic to a degree rarely seen in hog cholera. Superficially and on cross section the most hemorrhagic nodes resemble he matomas more than lymph nodes. Most consistently and severely hemorrhagic are the gastric, periportal, renal, and mesenteric nodes. The thoracic and mandibular nodes, which are less severely involved, are usually mottled with hemorrhage. In the superficial groups of nodes the hemorrhages are relatively slight and peripheral in distribution. An occasional node shows nothing but swelling, and the cut surface is moist. In a few cases the nodes are congested rather than hemorrhagic and appear diffusely pink to red. A grayish mottling of the peripheral or cut surface may indicate necrosis.

Histologically, the lymph nodes of swine which die or are killed in the terminal stages of African swine fever invariably present hemorrhage, necrosis, and reactive changes in capillary walls. Individual nodes do not necessarily show every one of these changes, but all three have been demonstrated in every case examined.

Small focal hemorrhages appear first in the subcapsular reticular spaces beneath the lymph sinuses and adjacent to the lymphoid follicles. As they increase in size, they progressively involve additional reticular tissue and lymph sinuses (Fig 8.3), and finally they infiltrate or displace the lymphoid follicles (Fig. 8.4). Hemorrhage is sometimes so severe that it obscures other changes.

Necrosis of lymphoid tissue, manifested by fragmentation and scattering of the nuclear chromatin of lymphocytes (karyorrhexis), is a prominent and characteristic feature (Figs. 8 5 and 8 6). Although most striking in the lymph nodes, it may, and usually does, occur any place in the body where lymphoid tissue is found.

As a result of the marked depletion of lymphocytes, the arterioles and capillaries are conspicuous and appear to be abnormally numerous They are contracted and empty, their walls thickened and edematous, and the endothelial cells are piled up and protrude into the lumina. In more advanced cases, the walls of small vessels, especially near areas of lymphoid necrosis, are often greatly thickened with a subendothelial accumulation of acidophilic, finely granular proteinaceous material containing some erythrocytes. Occasionally these altered vessels are thrombosed.

FIG. 8.3—Congestion and hemorrhage in a lymph node. A. Hemorrhage in a paratrabecular sinus. B. Lymphatic follicles are still present. C. There is no hemorrhage in the peripheral cell-poor tissue. Hematoxylin-eosin. X 22.
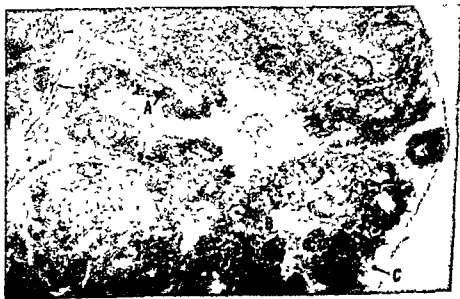
may occur. Cyanotic areas on the extremities are often noted.

The changes in the blood during the clinical course of African swine fever are similar to those of hog cholera. DeKock *et al.* reported an average drop of about 50 per cent in the total leukocyte count in 3 of 5 cases. The decrease in lymphocytes was commensurate. Blood counts have been reported for very few animals but leukopenia with an absolute lymphopenia would be expected from the extensive necrosis of lymphocytes observed histologically in lymphoid tissues. DeTray and Scott (1957) noted leukopenia beginning on the day the temperature rises, with the leukocyte count dropping steadily to 40 per cent of normal by the fourth day. They also observed an increase in the number of immature neutrophils.

Death usually occurs by the seventh day after onset of fever, frequently only a day or two after observance of the first signs of illness. The mortality rate invariably exceeds 95 per cent and usually approaches 100 per cent. The few recoveries reported have usually been among animals infected by contact with wild pigs. Establishment of the virus in domestic swine so enhances its virulence that recovery from African swine fever is rare in either natural or experimental infections. The few survivors may carry the virus for many months.

PATHOLOGY

General. Animals with the typical disease usually die so rapidly that loss of condition is uncommon. Early rigor mortis and rapid autolysis indicate the need for prompt necropsy examinations.

Cyanosis of the relatively hairless skin on the ears, snout, axilla, flanks, vulva, tail, and fetlocks is often striking in light-colored pigs. The cyanotic areas are reddish purple and sharply demarcated. The cyanotic dependent portions of the ears are frequently swollen. Discrete hemorrhages with dark centers and fading edges are seen grossly in the skin, particularly on the legs and abdominal wall.

Microscopically, small blood vessels in the dermis, especially the papillary dermis, are severely congested and account for the cyanosis seen grossly. The vessels are often occluded by fibrinous thrombi, and numerous eosinophils surround these vessels. Small necrotic lesions in the basilar layer of epithelium have been seen overlying the thrombosed vessels at the margin of cyanotic parts of the ear (Fig. 8.2).

When the thoracic and abdominal cavities are opened, the pericardial, pleural, and peritoneal fluids are excessive in quantity; they are clear and yellow and may be tinged with blood. Engorgement of vessels, especially small superficial vessels of the abdominal viscera and mesentery, is striking. Small tan to bright-red petechiae, so-called bran flecks, may be seen on the serosa of the viscera, especially of the small intestine. Dark hemorrhages deep in the wall of the colon are often conspicuous, and occasionally diffuse hemorrhage surrounds the kidney. Localized areas of edema may be found in the sublumbar, gastrohepatic, and
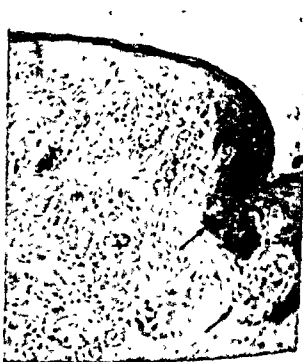
FIG. 8.2—Skin of the ear. Congestion of capillaries in the papillary dermis and necrosis of rows of the basilar layers of the epidermis. Hematoxylin eosin. X 175

interlobular septa 2 or 3 mm in width and by subpleural thickening both of which are most apparent on the pleural surface. Scattered petechiae and ecchymoses are found on the serous surfaces and in the parenchyma of the lungs in nearly every case. Viewed microscopically these are seen to be foci of interstitial congestion with or without interstitial hemorrhages and infrequently with alveolar hemorrhages (Fig 8 10).

In some cases karyorrhexis of lymphocytes similar to that in other lymphoid tissue occurs in parabronchial lymphoid nodules. Bronchopneumonia as a secondary complication is rare presumably because of the brief fatal course of the disease. Small areas of pre existent atelectasis and consolidation in the region of the cardiac notch similar to those encountered in swine in the United States are commonly found in European breeds of domestic swine in Africa.

Circulatory system There is often an excess of pericardial fluid. In a few instances it is cloudy and contains strands of fibrin. Cardiac hemorrhage of some degree is observed in about 70 per cent of the cases. Subepicardial and subendocardial hemorrhages are most common and are sometimes diffuse very extensive and striking. The subepicardial hemorrhages
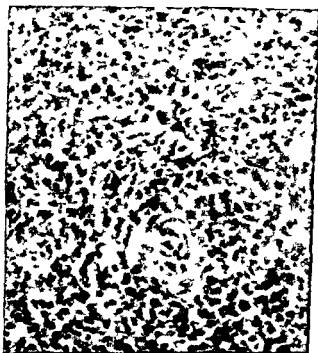
FIG 8 7—Splenic follicle in which adult lymphocytes are reduced surrounded by an accumulation of nuclear debris. Hematoxylin eosin X 270
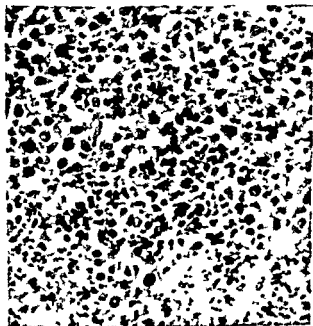
FIG 8 6—Higher magnification of the lymph node shown in Fig 8 5. Severe pyknosis and karyorrhexis of lymphoid cells. Hematoxylin eosin X 295

FIG 8 8—Spleen. Marked depletion of adult lymphocytes and effacement of ellipsoids. Hematoxylin eosin X 56

Spleen The majority of the spleens from animals with African swine fever are grossly normal Severe splenic engorgement with enlargement commonly noted in South African cases by Steyn (1928) DeKock *et al* (1940), and others was seen in only about 6 per cent of the 83 experimental animals infected with the East African strain of virus by Maurer *et al* (1958) When engorgement occurs it may involve only a portion of the organ In swollen areas the pulp is deep purplish black and it bulges from the cut surfaces Lymphoid follicles are few and small Small dark red raised triangular infarcts occurred along the edge of the spleen in about 7 per cent of the swine in the series studied by Maurer and co workers Engorgement of superficial vessels in the capsule is striking in some cases but its significance is questionable

Microscopically the usual changes are marked depletion of lymphocytes perifollicular and paratrabecular congestion characteristic necrosis and effacement of

FIG 8 5—Lymph node with extensive necrosis and loss of follicular architecture Hematoxylin eos n X 70

ellipsoids and reactive changes in the vessels similar to those in the lymph nodes (Figs 8 7 8 8 and 8 9) Massive karyorrhexis of lymphoid cells is frequent.

Respiratory tract The larynx particularly the epiglottis usually bears petechiae or ecchymoses In some instances it is extensively congested and presents more severe diffuse hemorrhages than occur in hog cholera Petechiae sometimes appear in the anterior third of the trachea Frothy fluid is likely to be present in the trachea when there is pulmonary edema

A slight excess of straw colored fluid is commonly seen in the thoracic cavity It is proportionately greater in the presence of pulmonary edema This edema is marked by broadened yellow gelatinous

FIG 8 4—Lymph node Most of the lymphoid tissue has been replaced by massive hemorrhage Hematoxylin eos n X 20

(Fig 8 12)   Karyorrhexis of the lymph ocytes usually is conspicuous and a char acteristic feature   The hepatic cells border ing on areas of cellular infiltration are com pressed and often necrotic   Necrosis how ever, is not confined to liver cells at these sites but occurs in individual cells scat tered at random throughout the lobules   Mild congestion is usually present

Gall bladder   Engorgement of superfi cial blood vessels in the wall of the gall bladder is the most striking change seen grossly   The wall may be edematous at times the edema extends to the liver cap sule, making it appear thickened and gelat inous along the line of attachment of the gall bladder   Petechiae and ecchymoses may be scattered over the serous or mu cosal surfaces   The gall bladder is usually distended with bile   Microscopically vas cular engorgement   submucosal edema karyorrhexis of lymphocytes in the sub mucosa and small hemorrhages are ob served   In some instances autolytic changes in the gall bladder wall may cause con densations of chromatin in nuclei of both epithelial and mesenchymal cells to give

the appearance of intranuclear inclusions (Fig 8 13)   These nonspecific inclusions are formed whenever there is sufficient autolysis of these cells therefore they may be observed in other conditions including hog cholera

Pancreas   In the cases studied by Maurer et al (1958) the pancreas usually appeared normal   In some instances ec chymoses were observed but not the severe hemorrhage and extensive necrosis re ported by DeKock et al (1940) in their larger series of cases in South Africa

Urogenital system   Hemorrhages most commonly petechiae occur in the kidney in about two thirds of the cases   After removal of the renal capsule careful exam ination will usually reveal a few scattered petechiae but they are seldom as numer ous as in some cases of hog cholera   If there are any subcapsular petechiae cross section will usually reveal more numerous petechiae in the cortex and medulla   In about 5 per cent of the cases studied by Maurer et al (1958)   very severe diffuse

FIG 8 12—Infiltration of the interlobular con nective tissue of the liver by lymphoid cells karyorrhexis of lymphocytes and necrosis of in dividual hepatic cells (arrow)   Hematoxylin eosin × 250

FIG 8 13—Autolytic condensation of nuclear chromatin in the gall bladder giving the ap pearance of inclusion bodies   Hematoxylin eosin × 300
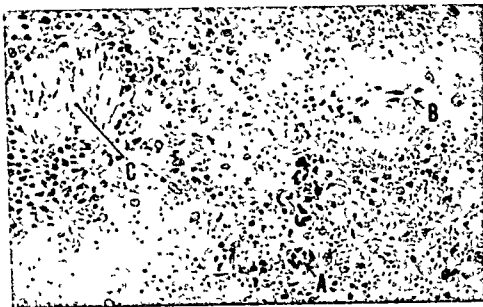
FIG. 8.9—Higher magnification of the spleen in Fig. 8.8. A. Central artery. The splenic follicle is devoid of lymphocytes. B. Lumen of an ellipsoidal vessel. The cellular sheaths of the three ellipsoids have been replaced by an acellular, finely granular, proteinaceous material in which a few erythrocytes are suspended. C. Segment of trabeculus. Hematoxylin-eosin X 235.

are concentrated around the major coronary vessels and are most extensive over the left ventricle. In a few instances some edema is seen in the tissues involved by hemorrhage. Subendocardial hemorrhages are also most severe in the left ventricle. The myocardium usually appears normal grossly, but interstitial hemorrhages, a few thrombi, and some areas of myocardial degeneration have been seen on microscopic examination (Fig. 8.11).

Liver. In African swine fever, the liver usually appears normal on gross examination. Mottling, with dark areas of congestion, is the most common abnormality, and in some cases tissues adjacent to the gall bladder are congested and edematous. Microscopically, the interlobular connective tissue is uniformly and strikingly infiltrated with lymphoid cells and smaller numbers of plasma cells and histiocytes
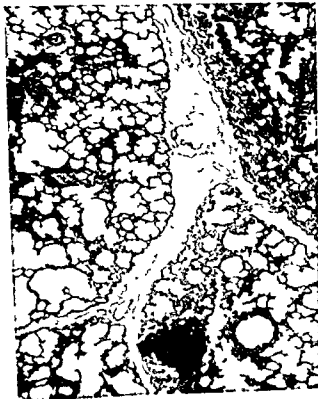
FIG. 8.10—Lung. Interlobular edema and one focus of congestion and hemorrhage. Hematoxylin eosin. X 17½.

FIG. 8.11—Heart. Subendocardial hemorrhage. Hematoxylin-eosin X 120.

FIG 8 14—Extreme congestion and edema in the submucosa of the colon Hematoxyl n eosin X 26½

ination Extreme engorgement of the large thin walled vessels in the submucosa is most striking (Fig 8 14) Congestion in the lamina propria and edema of the submucosa are also common

Central nervous system Congestion of the meninges is frequent but usually mild and small perivascular hemorrhages may be present (Fig 8 15) The brain itself appears normal grossly

Microscopically in addition to congestion and perivascular hemorrhages there is often a mild lymphoid infiltrate in the leptomeninges These lymphoid cells may undergo karyorrhexis as do those in the lymphatic tissues Tiny hemorrhages although few in number, are widely scattered throughout the brain especially the brain stem In contrast to the report of DeKock *et al* that perivascular round cell in filtrations were conspicuous by their ab sence Maurer *et al* (1958) found striking perivascular and intramural infiltrations in most cases although only a few vessels might be affected in any individual case (Fig 8 16) Often the vessel wall itself was distended with nuclear debris and

lymphoid cells while the Virchow Robin space appeared empty (Fig 8 17) Peri vascular infiltration of lymphoid cells was found regularly in the choroid plexus along with destruction of these lymphoid cells thrombosis and thickening of capil lary walls Generalized, early, acute neuro

FIG 8 15—Perivascular hemorrhage in the lep tomeninges Hematoxylin eos n X 50

hemorrhage was found in the walls of the calyces, and the pelvis was filled with blood. When severe diffuse hemorrhage occurred in the hilum, diffuse hemorrhages were likely to be seen beneath the capsule. In some cases, similar hemorrhages involving the perirenal fascia have been striking.

In general, the histologic changes in the kidney support the gross observations, but many lesions that resemble petechiae are actually foci of interstitial engorgement.

The adrenals usually appear normal, but occasional hemorrhages may occur in the cortex and medulla.

In most instances the urinary bladder appears normal. In about 30 per cent of the cases of Maurer et al. (1958), petechiae were seen on the mucosal surface; in about 20 per cent the mucosal surface was moderately and diffusely reddened.

The testes appear grossly normal but in some cases the epididymis is severely engorged.

Stomach. The stomach is usually filled with ingesta, thus attesting to the delayed anorexia. In about one half the cases there is evidence of acute, diffuse, often hemorrhagic, gastritis, which is most severe in the fundus. The hemorrhages vary from petechial to diffuse, with bleeding into the lumen. Ulcers, often covered with necrotic debris, appear in the pyloric and fundic regions in about one-fourth of the cases.

Small intestine. Inflammation of varying degree occurs in the small intestine in about two thirds of the cases, but these changes are generally less severe than in other parts of the digestive tract. The manifestations of enteritis vary from localized red areas, with petechiae on the crest of mucosal folds, to generalized severe inflammation and diffuse hemorrhage. Erosions are not observed. Occasionally petechiae and ecchymoses are scattered in the subserosa. In a few cases the Peyer's patches are visible on the serosal surface as yellowish edematous areas sprinkled with petechiae. The mesenteric blood vessels of the small intestine are consistently engorged and very conspicuous.

The region of the ileocecal valve normally contains mucus-filled crypts and numerous lymphoid nodules. Congestion and even ulceration of the mucosa of the ileocecal valve is so frequent in pigs with a variety of diseases that their presence in African swine fever is of questionable differential significance; however, infarction and the destruction of lymphocytes by karyorrhexis are typical of African swine fever.

Cecum. Significant changes are seen in the cecum in about 50 per cent of the cases. The lesions vary from mild reddening to severe hemorrhage, with ulceration of the mucosa. Petechiae along with blotches and longitudinal streaks of diffuse hemorrhage are most common. Lesions simulating the "button ulcers" of hog cholera are unusual and occur in the cecum or colon only in cases of long standing. When ulcers develop, they are usually small, deep, and covered with necrotic debris. Microscopically the ulcers appear to be the result of infarction. There is coagulation necrosis of the entire mucosal layer which is sharply demarcated from the adjacent normal-appearing tissue by a narrow zone of congestion, thrombosis, and degenerating cells. There is little or no cellular infiltrate, and bacterial colonies occur predominantly in the surface layers of necrotic debris. Subserosal hemorrhages are present in a few cases.

Colon. Inflammation of the colon occurs in about 50 per cent of the cases. It varies in severity, being seen as reddening, multiple petechiae or extensive lesions made up of confluent ecchymoses. The small ulcers that form infrequently in the colon are like those in the cecum. In many cases severe engorgement, which grossly resembles hemorrhage, occurs deep within the wall and is equally evident from either the mucosal or serosal surface. Ecchymoses may be seen on the serous surface. Edema commonly occurs in the submucosa and may extend into the mesentery, where it produces gross gelatinous thickening and increased transparency.

Gross congestion, hemorrhage, and edema are confirmed by microscopic exami-

If these lesions are found, especially in animals believed immune to hog cholera, the outbreak should be reported to state and federal authorities concerned with the control of animal diseases, and efforts made to confirm the diagnosis. In the absence of a serologic test and the lack of potent immune sera against the possible strains of African swine fever virus, a confirmatory diagnosis depends in part on the elimination of other diseases, especially hog cholera. A distinction from cholera can be made by challenging pigs known to be cholera immune with the suspect virus or less desirably, by demonstrating the inability of anti hog cholera serum of known high titer to protect test pigs against the suspect virus. African swine fever also can be diagnosed by histological study of the typical lesions which are described in this chapter and given in greater detail by Maurer et al (1958). The most characteristic lesions are the severe hemorrhages found throughout the body and marked karyorrhexis of lymphoid cells. The microscopic lesions of greatest diagnostic significance in African swine fever are in the lymph nodes, spleen, liver brain, and large intestine. Microscopic lesions that may facilitate diagnosis appear with less consistency in the kidney, gall bladder, lung, and heart. Impression smears of lymph nodes may reveal severe karyorrhexis of lymphocytes. When the diagnosis is to be made in a region where the disease has not occurred previously, the combined use of all methods of arriving at a definite diagnosis is recommended.

It is obvious that research is urgently needed to obtain information on the antigenic characteristics of each strain of African swine fever virus, to provide antisera for strain typing, and to develop a serologic test to confirm the diagnosis.

IMMUNITY

In view of the demonstrated ability of African swine fever to infect consistently and kill essentially 100 per cent of exposed domestic swine, it is evident why so little information is available on the immunity of recovered animals. In the few instances in which DeKock et al (1940), Montgomery (1921), and Steyn (1928) reported the experimental challenge of recovered animals, the immunity was found to be incomplete, inconstant, and transient even when the challenge was made with the homologous strain. DeKock et al noted that the virus persisted in the blood of recovered domestic swine for periods as long as 10 months, but the immunity status of these carrier swine has not been reported.

Many workers have demonstrated that swine immune or hyperimmune to hog cholera are susceptible to African swine fever.

To date, numerous attempts to develop a useful vaccine against African swine fever have failed. Methods used to inactivate the virus for vaccine production have included heat, Lugol's solution, formalin, toluol (Walker, 1933), and crystal violet (DeKock et al, 1940). In one instance Walker found that immunity conferred by the immune serum virus simultaneous method still existed at 192 days but not at 283 days. He also observed that animals immunized by the serum virus method, and then hyperimmunized by additional doses of the same virus, were susceptible when exposed to virus from another source (strain). Methods involving the use of immune sera would be impractical because of the difficulty in preparing immune sera.

South African workers have reported that pigs inoculated with virus partially attenuated by egg passage survived and were subsequently immune to the original homologous South African strain but not to a strain from the Belgian Congo.

PREVENTION AND CONTROL

Prevention and control are presently dependent upon an awareness of the ever present threat of African swine fever, import restrictions, prompt diagnosis, quarantine, and slaughter. The need for the development of a diagnostic serologic test and an effective vaccine is obvious and urgent.

nal degeneration was observed in all brains studied, even when other changes were few. Sometimes the neuronal degeneration was associated with neuronophagia and focal glial proliferation.

DIAGNOSIS

The major problem in the diagnosis of African swine fever is that of differentiation from hog cholera. The clinical features and gross lesions of African swine fever are so similar to those of hog cholera that careful observation, examination, and necropsy of several animals from a suspect herd are needed to distinguish one from the other. These studies should not be limited to one animal, for the gross lesions in individual cases of either hog cholera or African swine fever may be meager and inconspicuous. *The diagnosis is best made on a herd basis and should always be supported by histopathologic study or the inoculation of hog cholera-immune animals, preferably both.* The only animal suitable

FIG. 8.17—Brain. Infiltration of a vessel wall, with karyorrhexis and pyknosis of the infiltrating lymphoid cells. Hematoxylin-eosin. X 305.

for diagnostic inoculation is the domestic pig.

African swine fever should be suspected when, in a febrile disease of swine, other clinical signs frequently fail to develop until within 48 hours of death, and when the disease is cholera-like, highly contagious, essentially 100 per cent fatal, and productive of gross lesions similar to, but more severe than, those of hog cholera. The most significant gross lesions include: cyanosis and hemorrhages of the skin, hemorrhages of the lymph nodes, hemorrhages on the heart and kidneys, pulmonary interlobular edema, and congestion and edema of the gall bladder.[1]

FIG. 8.16—Brain. Marked infiltration of the vessels and perivascular spaces by lymphoid cells. Hematoxylin-eosin. X 140.

[1] A movie entitled "Hog Cholera and African Swine Fever, A Comparison," illustrating the gross lesions present in the series of cases described here in by Maurer et al. (1958), has been prepared by the joint efforts of the Armed Forces Institute of Pathology and the USDA. It may be obtained from the Motion Picture Service of the USDA.

A study set consisting of 50 color transparencies of the gross and microscopic lesions and 25 tissue sections from the same cases is available from the Armed Forces Institute of Pathology.

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Pox in Swine

Pox in swine has been reported in Europe, Asia, Africa, and North and South America It occurs in practically all areas in which the swine population reaches proportions of economic significance This infection reaches its highest incidence in the mid western area of the United States, where the hog population is the greatest

The earlier studies and reports of pox in swine were recorded in Europe Spinola (1842) reported on investigations of pox infection in swine and expressed serious doubt as to the existence of a primary pox in swine The virus strains used in his studies appeared to be closely related to those of vaccinia Peiper (1901) believed that human variola could be transmitted to swine and that it was possible to infect man with swine pox virus Poenaru (1913) transmitted pox infection from diseased to healthy young pigs and found that the disease was caused by a filtrable virus There is definite evidence from the studies conducted by some of the more able pathologists that the pathologic changes in swine characteristic of pox, may be produced by two different viruses Nearly all of the pox infection studied by Yoshikawa (1930) and Akazawa and Matsumura (1937) involved virus strains that were immunologically similar to that of vaccinia Akazawa and Matsumura were able to establish typical infections in rabbits with these virus strains Velu (1916) was

not able to establish the viral infection from swine to rabbits McNutt et al (1929) likewise failed to infect rabbits with swine pox virus Manninger and his associates (1940) reported an outbreak of pox in swine from which vaccinia virus was iso lated Manninger as well as other workers described a disease of swine characterized by pox lesions caused by a filtrable virus immunologically different from vaccinia virus This virus could not be transmitted to rabbits The gross and microscopic pathology of the pox lesions produced by these two types of virus are strikingly similar Schwarte and Biester (1941) con ducted extensive investigations on pox in fections in swine with special emphasis on host species susceptibility and immunolog ical relationships The experimental data secured in these studies clearly indicated that typical pox infections in swine may be produced by vaccinia virus as well as virus strains infectious only to swine

TERMINOLOGY OF POX INFECTIONS IN SWINE

The majority of the cases of pox in swine studied in this country and abroad were associated with virus that was immunolog ically similar to vaccinia Likewise, the virus could be established in susceptible hosts other than swine Manninger et al (1940) proposed that swine pox be used to designate the disease produced in swine

REFERENCES

DeKock, G Robinson, E M, and Keppel, J J G 1940 Swine fever in South Africa Onderstepoort Jour Vet Sci and Anim Ind 14 31

DeTray, D E and Scott, G R 1957 Blood changes in swine with African swine fever Amer Jour Vet Res 18 484

Henning, M W 1956 Animal Diseases in South Africa, 3rd ed Johannesburg, South Africa Central News Agency Ltd, p 871

Malrer, F D, Griesemer, R A, and Jones, T C 1958 The pathology of African swine fever, a comparison with hog cholera Amer Jour Vet Res (in press)

McIntosh, B M 1952 The propagation of African swine fever virus in the embryonated hen s egg Jour South Afr Vet Med Assn 23 217

Montgomery, R E 1921 On a form of swine fever occurring in British East Africa (Kenya Colony) Jour Comp Path and Therap 34 159

Steyn, D G 1928 Preliminary Report on a South African Virus Disease Amongst Pigs 13th and 14th Reports Director of Vet Ed and Res, Onderstepoort, South Africa p 415

Velho E L 1956 Observations sur la peste porcine en Angola (African swine fever in Angola) Bull Off int Epiz 46 335 Abstr Vet Bull No 3804, 46 625 1957

Walker J 1933 East African Swine Fever Thesis Univ Zurich, Ballière, Tindall and Cox London

VACCINIA (Variola vaccina, Cow Pox)

This type of pox virus has a wide variety of hosts, and the infection may be transmitted to man as well as to practically all domesticated and laboratory animals The majority of outbreaks of pox in swine are caused by vaccinia virus infection The greatest incidence occurs in young swine which are highly susceptible to infection Older swine are quite resistant while the mature swine retained as breeding stock rarely contract the disease Some authorities have suggested the possibility of mature swine having recovered from pox infection at an early age, thereby developing an active immunity

The incubation period the course of the disease through the various stages and the pathologic condition produced by vaccinia infection in swine are identical with those produced by the swine pox virus (For more detailed information on vaccinia consult Smadel, 1948) Pox infection in swine and the relationships of the two virus infections will be described in more detail under swine pox '

Swine recovering from vaccinia infection develop strong immunity for life The immunity is specific for vaccinia only and not for swine pox infection

SWINE POX (Variola suilla)

NATURAL INFECTION

Young swine are susceptible to infection by swine pox virus while the older and mature animals are highly resistant, which is likewise applicable to most animal pox diseases The majority of outbreaks of swine pox under field conditions, un complicated by other diseases, are quite similar in nature The disease is usually confined to suckling pigs ranging in age from 1 to 6 weeks The lesions are predominantly located on the backs and sides of the affected animals Rarely, individual pigs may develop generalized pox eruptions, including lesions about the nose and

mouth associated with febrile reactions loss of appetite, and progressive weakness usually terminating in death The typical lesions measure approximately one centimeter in diameter, are round and rather smooth, with no areas of depression The margins of these lesions become light gray similar in appearance to lesions frequently observed in some fungous infections (Fig 9 1)   Dark brown crusts develop shortly after the maximum development of the lesions There appears to be considerable skin irritation during the course of the disease Consequently, the infected swine resort to scratching and rubbing on wire fences buildings, and other equipment causing the integument to crack and break and allowing serous or hemorrhagic exudate to be liberated from the skin lesions The exudate attracts flies and insects causing secondary infection, and in all probability these insects are responsible for the spread of infection to other susceptible animals in the immediate vicinity In one particular swine herd under observation no lice could be found on any of
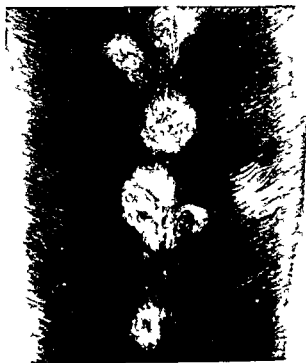
FIG 9 1—Swine pox lesions on the back and sides of experimental swine

by the virus immunologically similar to vaccinia virus and that the identical pathologic condition produced by the filtrable virus, proved to be immunologically distinct from that of vaccinia, be designated as poxlike eruptions of swine This proposal, if accepted, would only add confusion to a rather unusual pathologic complex Schwarte and Biester (1941) have clearly demonstrated through cross immunity studies and species susceptibility tests that virus strains which for years have been regarded by scientists as "similar" to vaccinia are *identical with vaccinia and should be regarded as vaccinia infection in swine* The pox infections caused by virus strains immunologically distinct from vaccinia and which can be transmitted to swine only, should be designated as swine pox *These designations are definite and eliminate the confusion in terminology which has existed for years*

ETIOLOGY

Pox infections in swine are caused by *filtrable viruses* The general properties of the pox viruses are quite similar According to Stanley (1947) the pox viruses range from 210 to 260 mμ in size The viruses are dermotropic in nature and their activities are largely confined to epithelial tissue Pox infections in swine may be spread by the hog louse (*Haematopinus suis*) which is a rather common blood sucking external parasite Occasionally, a septicemic form of the disease will appear in an individual animal, which terminates fatally in most cases The host range varies greatly Vaccinia may be transmitted to a wide variety of animal species while such viruses as avian and swine pox are specific for their respective susceptible avian and porcine hosts These viruses are filtrable and will pass through the Berkefeld V and N filters as well as the Chamberland L$_3$ filter A considerable amount of virus adheres to the filters during the filtration process Many of these pox viruses may be cultivated on artificial media in the presence of living cells

VACCINIA VIRUS

Vaccinia virus has been studied in greater detail than any of the pox viruses and consequently more is known of the characteristics and the general properties of the virus Smadel (1948) indicates that the virus particle is of the order of 236 mμ in size The virus is filtrable through Berkefeld V, N, and W filters as well as Chamberland L$_3$ There is considerable adsorption of the virus to most filters Vaccinia virus exposed to 55° C for 20 minutes is destroyed Virus particles incorporated in dried crusts remain active for more than one year and may be stored in glycerin for a longer time Exposure to ultraviolet light destroys the virus rapidly Various commercial viricidal agents are destructive to vaccinia virus The virus of vaccinia can be cultivated readily *on all susceptible laboratory and domesticated animals* It has been successfully grown on *the scarified cornea of the rabbit, on the chorio-allantoic membrane of the chicken embryo, and on suitable tissue culture media*

SWINE POX VIRUS

Comparatively little research has been done on the characteristics and physical properties of swine pox virus The virus passes through Berkefeld V and N filters It also has been filtered *through the* Chamberland L$_3$ porcelain filter Filtration studies indicate that the virus of swine pox approximates the size of other animal pox viruses which range from 210 to 260 mμ We have no confirmation of the virus size by the filtration techniques used *Ultrafiltration through membranes of graded porosities, ultracentrifugation, and sedimentation constants have not been used in the study of swine pox virus* The virus is host specific for swine Virus incorporated in the dried crusts, or in glycerin at room temperature, or under refrigerated conditions remains active for a long time Like vaccinia virus, it may be destroyed by adequate exposure to sunshine or *ultraviolet light*

instances Subsequent inoculation of re covered swine failed to produce any re action, indicating a well developed im munity

SPECIES SUSCEPTIBILITY

Experimental inoculations of swine pox virus were made into various species of animals including the horse calf, sheep pig, dog, cat, domestic fowl, rabbit, guinea pig, rat, mouse, and man to determine the susceptibility of these species to this viral infection Cutaneous inoculations were made, similar to those previously described for successful transmission of swine pox to susceptible swine The first inoculations were made with the Berkefeld N filtrate The pig was the only species in which posi tive reactions were produced Two weeks after the first inoculations were made, these same animals were inoculated with crude unfiltered viral suspension No re actions resulted from these inoculations The susceptibility tests in man were con fined to the use of the Berkefeld V and N filtrates to avoid possible bacterial infec tion from the crude viral suspension No positive reactions were obtained

IMMUNOLOGICAL RELATIONSHIP OF SWINE POX VIRUS AND VACCINIA VIRUS

The problems associated with pox in fections in swine herds under field condi tions create considerable confusion to the veterinarians as well as the swine pro ducers It might be well to briefly review some conclusive experimental evidence to clear up questions on immunity involving swine pox virus and that of vaccinia The extensive researches carried on with vac cinia virus over a period of years have clearly proved that various species of animals are more or less susceptible to vaccinia infection, particularly the rabbit which is highly susceptible to this disease Because none of the species exposed, with the exception of the pig, showed a positive reaction to inoculation with the swine pox virus, it was desirable to determine whether or not these animals were immune to vaccinia Experimental animals includ

ing the horse, calf, and rabbit, which failed to react to the inoculation of swine pox virus, and a number of pigs which had recently recovered from the swine pox infection, were inoculated cutaneously with vaccinia virus Strong positive re actions were secured in the horse, calf, pig, and rabbit inoculated with the vac cinia virus The reactions in the sheep and dog were definitely positive but not nearly as severe as those of the other animals Since the experimental animals which were refractory to infection with the swine pox virus proved to be suscep tible to that of vaccinia it would indicate that the viruses are immunologically dis tinct Furthermore, the positive vaccinia reaction in the pigs which had recently re covered from swine pox indicates that there was no immunity or protective influence developed against vaccinia infection How ever, pigs recovering from swine pox infec tion proved to be immune to subsequent inoculation with swine pox virus Based on the experimental evidence presented there is no immunological relationship apparent between the virus of swine pox and that of vaccinia

PATHOLOGY

In field cases the lesions were largely confined to the backs and sides of the dis eased animals The early lesions developed following hyperemia and lymphatic dis tention to form papules as the result of epithelial proliferation This proliferation extended into the deeper structures in volving the stratum granulosum The de generative process observed in these lesions was not as extensive or severe as usually encountered in typical pox lesions The superficial, necrotic, epithelial layers de veloped into smooth grayish brown crusts which in many instances became cracked, exuding serous matter The stages of vesiculation and pustulation were not as well defined as those commonly en countered in pox lesions Regeneration of the epithelium, accompanied by desiccation of the necrotic tissue and crust formation, was rapid Extensive leukocytic infiltration

the swine on the farm. The swine were confined in unit areas of one acre in size. The infection spread rapidly to four of these units in one section of the farm. Later, the disease appeared in two units about one-quarter mile from the original focus of infection. The animals in other units were not affected. The disease spread rapidly in the infected areas until the arrival of cold weather which arrested the activity of the flies and mosquitoes. The course of the disease was about 6 weeks in most cases. Delayed recovery as the result of secondary infection was observed in a number of instances. The mortality in uncomplicated swine pox is negligible, but seriously retarded growth and development are usually observed.

TRANSMISSION

Swine pox virus for use in transmission experiments (Schwarte and Biester, 1941) was secured from well-developed lesions on naturally infected swine raised and maintained under field conditions. This material was carefully selected from lesions which showed no evidence of secondary bacterial infection. The material was finely triturated in a mortar to which physiological salt solution was added. A thousandfold dilution was made, and a portion of this crude viral suspension was filtered through Berkefeld filters. Both the filtrates and the unfiltered preparations were used for animal inoculation.

Healthy young pigs approximately 8 weeks of age were selected for inoculation. Suitable areas of the skin on the back and underline were thoroughly cleansed and disinfected following the removal of the hair. The residual disinfectant was washed off with sterile distilled water. Inoculations were made by means of needle punctures and the usual skin scarification technic. Crude, unfiltered viral suspensions and filtrates, passed through Berkefeld V and N filter candles, were used. Positive reactions developed in all inoculated pigs. A mild general reaction followed by a slight rise in temperature was observed several days after inoculation. Small red spots appeared at the point of inoculation 4 and 5 days later. The lesions developed rapidly into smooth, hard elevations, which ranged from 1 to 3 cm. in diameter, on the backs and sides of the inoculated swine. Hard crusts formed on the surfaces of these lesions, which soon cracked and dropped off. On the underline, small eruptions developed into typical pox lesions passing through the successive stages of papule, vesicle, and pustule formation, becoming umbilicated and covered with dark-brown crusts or scabs (Fig. 9.2). The scabs dropped off in several days, and complete recovery followed in 12 to 14 days in most

FIG. 9.2—Swine pox lesions on the underline of experimental swine.

the herd infection for considerable time
Herd infections occurring in late summer
may continue until cool weather arrests
the activity of flies and mosquitoes which
are a factor in the spread of infection,
especially where skin lesions coalesce and
are broken by rubbing or scratching
Secondary infections of these lesions delay
recovery On rare occasions, the disease
assumes a septicemic form accompanied by
a high temperature (107–108° F), weak
ness, loss of appetite, chills, depression,
and rapid dehydration Lesions may be
come generalized, extending to the eyes,
nose, and mouth, with eruptions some
times developing on the mucous mem
branes of the pharynx, trachea, and bron
chi Progressive weakness and respiratory
complications may be observed prior to
death

The true course of the disease, un
complicated by other conditions, can be
better observed under experimental con
ditions Small red papules appear 4 or 5
days after the virus is placed on the scari
fied skin A slight temperature elevation
and general reaction may occur at this
time The lesions may develop into abor
tive vesicles and pustules, or on the back
of the pig the lesion usually enlarge rapidly
into hard elevations from 1 to 3 cm in
diameter The hard crusts that form on
these areas drop off in a few days The
course of the disease usually runs from
14 to 20 days

DIAGNOSIS

An accurate diagnosis of pox in swine is
not difficult when skin lesions in the vari
ous stages of development can be ob
served on the underline of the infected
swine The lesions on the back and upper
sides can be confused with fungous infec
tions such as ringworm There are no
lesions found on the feet The disease
runs a definite course recovery is com
plete and is followed by an active im
munity Differentiation of swine pox from
vaccinia can be determined by cross-im
munization tests The vesicular lesions on
the mucous membranes of the mouth and
tongue together with the foot lesions of
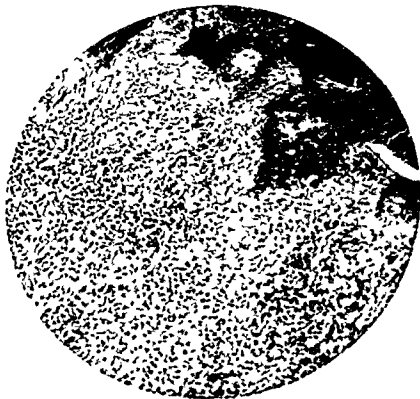
FIG 9 4—Degeneration and
suppuration X 150
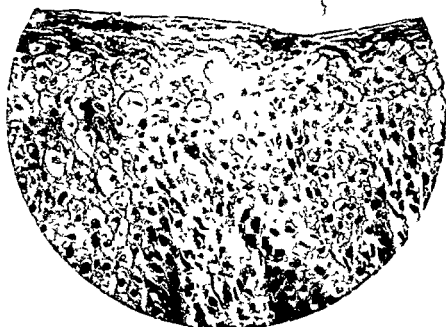
FIG 9 3—Leukocytic infiltration and cell vacuolization X 300

was commonly observed in areas involving secondary infection Regeneration of the epithelial structures was seriously retarded in areas where considerable necrosis of the epithelium occurred

The lesions on the back, in experimentally induced cases, were comparable with those described from field cases However, the experimental animals were confined to clean pens in a screened building comparatively free from insects Secondary infection of the lesions was eliminated The lesions which developed on the underline of the experimental swine were not characterized by an extensive cellular proliferation The localized hyperemia and congestion were more evident The vacuolization and distention of cells seemed to be greater The stages of vesiculation and pustule formation were more marked Desiccation seemed to take place first in the center of the lesion, causing a definite umbilication typical of the true pox lesion The epithelial regeneration was more rapid on the underline areas than on the backs of the experimental

swine The greater vascularity of the skin on the underline of the pigs undoubtedly plays an important part in the comparatively more rapid epithelial regeneration in this area

The histopathologic examination of these different types of lesions showed the same general changes, including the initial inflammatory reaction followed by degeneration of the epithelial cells, later infiltration with leukocytes, and finally the epithelial regeneration These lesions are characteristic of typical pox lesions

COURSE

In the great majority of cases, pox runs a typical and favorable course An outbreak of pox in a large swine herd frequently goes without detection until some of the infected animals rub and scratch on fence posts, feeders, buildings, or other equipment, as the result of the dermatitis caused by developing skin lesions Several weeks may elapse before all of the susceptible swine in the herd have become infected, which extends the duration of

around the hair follicles Bedding which contains certain irritating substances may produce considerable skin reaction Baby pigs farrowed and raised in the field often develop dermatitis from certain weeds or short stubble residues of grain fields The raised areas about the hair follicles are often confused with papule formations of swine pox They do not develop into vesicles pustules or scabs The dermatitis may persist for weeks especially if weather conditions are favorable for continued skin irritation After the animals reach forty or fifty pounds the skin becomes less sensitive and dermatitis of this type usually disappears Foot and mouth disease and vesicular exanthema are two diseases which should not be confused with swine pox Delayed diagnosis of either of these highly infectious diseases may result in a rapid spread of these infectious agents and a great economic loss through eradication by slaughter and quarantine

PROGNOSIS

Pox in swine usually runs a definite course and the prognosis is good in uncomplicated cases The losses are negligible and the only effects on the swine are retarded growth and development except in occasional individuals that develop the septicemic form of the disease which terminates fatally Other infectious diseases especially those which involve the respiratory tract may be complicating factors responsible for high mortality in pox infected swine Heavy losses following unfavorable reactions have been reported

FIG 9 6—Epithelial regeneration   X 150

FIG. 9 5—Scab formation following degenerative changes. X 150.

foot and mouth disease, are sufficient to differentiate this infection from swine pox.

The typical lesions of vesicular exanthema, including the foot lesions, should not be confused with the lesions of swine pox. Skin lesions on the underline of young pigs associated with hog cholera do not progress through the successive stages characteristic of swine pox lesions The clinical signs observed in pigs during the various phases of hog cholera are not manifested in swine pox infection. The skin lesions commonly observed in the course of swine erysipelas do not resemble those of swine pox. A viral disease of young swine reported by Durand *et al.* (1936) could be confused with the comparatively rare septicemic form of swine pox. This condition is sometimes referred

to in the literature as swineherd's disease because it is frequently transmitted to stockmen and farmhands from diseased swine. Young pigs develop a generalized reaction followed by a maculopapular rash and conjunctivitis Pigs which succumb to the disease show hemorrhages in the brain, throat, intestine, and renal pelvis. Animals susceptible to this infection include young swine, ground squirrels, ferrets, mice, and cats, as well as man. Swine pox is definitely a specific disease of swine and not transmissible to other species of animals or man.

Various forms of dermatitis are frequently confused with pox in man as well as swine. Young pigs have a rather sensitive skin and are subject to inflammatory reactions usually causing small raised areas

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CHAPTER 10

Vesicular Exanthema

Vesicular exanthema of swine (VES) is an acute, febrile, infectious viral disease, characterized by the formation of vesicles on one or more parts of the body The course of the disease is usually about 1 to 2 weeks, the mortality is low, and recovery following uncomplicated viral infection is complete The incubation period in both the natural and experimental diseases varies from 24 to 72 hours, with extremes ranging from 12 hours to 12 days All ages as well as all breeds of swine appear to be susceptible (Madin and Traum, 1955) Occult cases do occur (Mott *et al*, 1953, Bankowski *et al*, 1955)

HISTORY

On April 23, 1932, a disease afflicting only swine, and clinically indistinguishable from foot and mouth disease (FMD), was reported on a hog ranch near Buena Park, Orange County, California and inspection of the entire area was immediately instituted by state and federal authorities On April 28, infected swine were found on 2 additional ranches near by On April 30, the disease was discovered on 2 adjoining ranches at Bellflower, Los Angeles County, some 15 miles distant from the original Buena Park focus By May 4 the disease had spread to a third ranch nearby, and this was the extent of the infection as it appeared in Los Angeles County The disease was then discovered on a

ranch located about 2 miles north of the original Buena Park focus on May 3, thus ending the spread of infection in Orange County Inspection of a ranch in San Bernardino County, on May 5 and 6, showed the disease to be present although separated by 50 miles from the other two foci The San Bernardino County infection was the last to be reported and represents the known extent of the 1932 outbreak The disease was diagnosed FMD, all animals directly and indirectly involved in the outbreak were slaughtered and buried the premises washed with lye solution, and all new livestock excluded for 30 days In demnities of $203 328 for the loss of the 18 747 swine 46 cattle, and 24 goats were paid jointly by the state of California and the federal government (Madin and Traum, 1955)

The virus from the 1932 outbreak failed to induce lesions in 24 guinea pigs, 2 calves 2 heifers, 1 adult cow, and 2 horses On the basis of these tests the diagnosis of FMD was made even though Traum recognized that it was rather atypical (Traum, 1933, 1934) All virus collected during the outbreak was ordered destroyed

In March of 1933, a disease again restricted to swine and clinically similar to the 1932 outbreak appeared in San Diego County, California, 100 miles distant from the 1932 foci The original focus and the immediately adjoining ranch were both

subsequent to hog cholera vaccination of pox infected swine

IMMUNITY

Swine which have recovered from pox infections usually develop an active immunity for years Immunity from swine pox does not offer any protection against vaccinia infection Immunity following recovery from vaccinia gives no protection against swine pox It is possible to carry out protective vaccination with both swine pox and vaccinia virus Virus secured from the lesions of an infected animal can be used for cutaneous vaccination Young pigs vaccinated with glycerinated pox virus develop localized lesions without generalized reactions followed by immunity The majority of pox outbreaks in swine cause little loss aside from temporary retarded growth and development so vaccination is not usually needed or practiced to any extent in this country

REFERENCES

AKAZAWA S AND MATSUMURA T 1937 Rep Govt Inst Vet Res (Fusan) 10 8

DURAND P GIROUD P LARRIVE E AND MESTRALLET A 1936 Transmission expérimentale a l'homme de la maladie des porchers Compt rend Acad Sci 203 830

MANNINGER R CSONTOS J AND SALYI J 1940 Über die Ätiologie des pockenartigen Ausschlags der Ferkel Arch f Tierheilk 75 159

McNUTT S H MURRAY C AND PURWIN P 1929 Swine pox Jour Amer Vet Med Assn 74 752

PEIFER H 1901 Die Pockenimpfung p 20

PICKLES E G AND SMADEL J E 1938 Ultracentrifugation studies on elementary bodies of vaccine virus I General methods and determination of particle size Jour Exper Med 68 583

POENARU J 1913 Recherches sur le virus filtrant dans la variole des porcelets Bull Soc cent Med Vet 67 148

SCHWARTE L H AND BIESTER H E 1941 Pox in swine Amer Jour Vet Res 2 136

SMADEL J E 1948 In Rivers T M Viral and Rickettsial Infections in Man J B Lippincott Co Philadelphia pp 314 328

SPINOLA M 1812 Krankheiten der Schweine A Hirschwald Berlin p 204

STANLEY W M 1947 Chemical studies on viruses Chem and Engin News 25 3786

VELU H 1916 Contribution à l'etude de l'étiologie de la variole des porcelets Recueil Med Vet. 92 24

YOSHIKAWA M 1930 Die Untersuchung uber Schweinepocken in Mandschurei (2 Mitteilung) Jour Jap Soc Vet Med 9 2

Angeles County, with the largest hog population in the state, had been free of the disease for 6 years prior to March, 1940 (Hurt, 1940–41).

On December 4, 1939, an outbreak of VES was found on one raw-garbage-feeding hog ranch in San Mateo County. An immediate and rigid quarantine was imposed on the infected area Slaughterers, commission firms, and stock yard officials were ordered not to accept shipments of hogs from the infected area. This economic quarantine was relaxed only when a definite diagnosis had been made, and then only swine coming from noninfected premises could be slaughtered. In addition, all hogs going to slaughter from the area were individually examined. In spite of all the quarantine efforts, 223,000 hogs, on 123 premises located in 25 counties, became infected. Within 6 months, one fourth of the state's hog population was involved (Duckworth, 1953a).

From June to October of 1940, a respite from the disease occurred, but on October 5, 1940, the infection reappeared in 12 counties in the central portion of the state, and in December of 1940 it occurred in Los Angeles County, involving 57 premises and 54,250 additional swine. During the year 1940, 277,250 swine on 169 premises were infected. The 1940 outbreak was notable for its severity and for the inclusion of 7 grain-feeding ranches and one stockyard This was the first time that infections were observed on nongarbage-feeding premises (Duckworth, 1953a)

After 1940 the recording of individual outbreaks was discontinued; instead, the total number of outbreaks for any one calendar year was recorded. Since 1940, the disease has recurred in California each year through 1955 Table 10 1 shows the number of outbreaks, the origin, and the number of swine involved per year for the first 20-year period (1932–51), during

TABLE 10 1

INCIDENCE OF VESICULAR EXANTHEMA IN CALIFORNIA FOR THE PERIOD 1932 TO 1951, SHOWING NUMBER AND TYPE OF INFECTED PREMISES*

| Year | Number of Outbreaks According to the Types of Premises | | | Number Swine Involved | Total Swine in State | Per Cent Total Swine Infected |
	Garbage Feeding	Grain Feeding	Slaughter House			
						(%)
1932	5	0	0	18,747	672,000	3
1933	3	0	0	5,533	706,000	0 7
1934	31	0	0	95,917	660,000	14 4
1935	4	0	0	10,100	530,000	2 0
1936	14	0	0	13,625	610,000	3 1
1937	0	0	0	0	732,000	
1938	0	0	0	0	820,000	
1939	15	0	0	32,000	763,000	0 4
1940	161	7	1	277,250	885,000	31 3
1941	155	15	0	160,104	876,000	18 0
1942	15	0	0	84,300	894,000	0 9
1943	122	3	14	288,355	1,019,000	28 0
1944	154	7	10	429,876	1,060,000	41 5
1945	58	0	2	127,620	763,000	16 7
1946	52	0	1	108,732	717,000	15 2
1947	129	10	4	212,535	664,000	32 0
1948	25	0	0	84,566	641,000	13 0
1949	101	0	4	199,875	671,000	29 8
1950	169	6	9	272,222	687,000	39 7
1951	53	1	4	82,442	653,000	12 4
Totals	1,266	49	49	2,514,299	11,808,000	

* Modified from Madin and Traum (1955)

found to be infected on March 20, and the infection was reported from a third ranch on March 31, and from a fourth a few days later (Madin and Traum, 1955) Virus from this outbreak was collected and tested in a variety of animals Infection was established in all of 15 swine and in 4 of 9 horses, but in none of 7 cattle and none of 37 guinea pigs (Traum, 1934)

Similar results from a larger number of animals were obtained by Mohler (1933a), and Reppin and Pyl (1935) Observers of the animal tests, with experience in FMD, saw no definite points of clinical difference between that disease in swine and the one produced by the San Diego virus Since the animal tests permitted no official diagnosis, the usual slaughter and quarantine methods were invoked Indemnification in the amount of $45,350 was made for the slaughter of 5 578 animals (Duckworth and White, 1943, Mohler, 1933b)

Cross immunity tests with vesicular stomatitis virus (VSV, types Indiana and New Jersey) and FMD virus (types A, O, and C) showed that the San Diego virus was immunologically distinct In comparing the 1932 and the 1933 outbreaks, Traum (1934) concluded that

The true classification of the virus causing the 1932 swine outbreak of foot and mouth like disease must be considered as not having been definitely determined even though a diagnosis of foot and mouth disease has been made and eradication carried out accordingly It is believed if more horses had been used in the tests, that lesions would have been produced, thus making the virus of 1932 and 1933 alike in every respect.

Following the 1933 outbreak, a new disease of swine was described by Traum (1934) in the following statement

Thus we are confronted by a vesicular disease in swine, which so far has shown as much difference in experimental inoculations and immunological tests from both vesicular stomatitis and foot and mouth disease as does foot and mouth disease from vesicular stomatitis and although great similarity exists between the viruses of vesicular stomatitis and foot and mouth disease, we have been designating them as separate diseases It therefore seems that with the information

tion at hand the swine disease discussed above should be recognized as a new entity Vesicular exanthema of swine is suggested as a name for this disease

In June of 1934, fifteen months after the San Diego outbreak of 1933, the disease appeared on a raw garbage feeding hog ranch near San Jose, California, some 500 miles distant from the San Diego foci (Duckworth and White, 1943, Duckworth, 1953a) During the next 3 months the infection spread to 27 ranches in 5 counties in central California and to 4 ranches in Los Angeles and San Bernardino counties, 400 miles to the south A total of 31 premises and 95,000 hogs were affected All of these ranches practiced raw garbage feeding, and, again, only swine were involved Virus recovered from this outbreak regularly infected swine Horses were only occasionally susceptible, and cattle and guinea pigs were completely refractory (Duckworth and White, 1943)

In this outbreak, the usual slaughter program was not employed Instead, a rigid quarantine was imposed on infected premises until all evidence of the disease had disappeared Trucks used for hauling garbage were disinfected upon departure, and steps were taken to minimize contact between trucks, truck drivers, and ranch attendants with other hog ranches or live stock premises

In 1935, the disease reappeared on 4 of the premises infected in 1934 and involved about 13,000 hogs This time the disease was relatively mild, and the quarantine measures were again imposed (Duckworth 1953a) During 1936, the disease appeared first on April 8 on one ranch in San Diego County and infected approximately 90 per cent of the animals The infection did not spread to neighboring ranches but instead, on April 21, appeared in the San Francisco Bay area, 500 miles north of San Diego By June 20, 13 premises were involved No cases were reported between June 20, 1936, and December 1 1939, despite the fact that regular inspection of garbage feeding hog ranches was carried out (Duckworth, 1953a) Los

the C$_3$H/CRGL, C$_{57}$BL/CRGL hybrid black, and Namru No visible evidence of disease is produced by types A$_{48}$ and B$_{51}$ when inoculated intracerebrally into suckling mice (Madin et al, 1958a) Man apparently is not susceptible

Madin and Traum (1953) reported that the hamster could be infected with the 1940 A and B strains if the inoculations were made intradermally over the abdomen Vesicles were formed at the site of inoculation within 24 hours and were accompanied by a significant pyrexia The vesicles ruptured soon after formation and no further reactions were visible Inoculation of hamsters with the current A$_{48}$ and B$_{51}$ strains gave completely negative results It is assumed that sufficient differences exist among the various strains of the virus as had been suggested by Crawford (1937), to account for the varying degrees of success with this particular host In addition Madin (1956a) failed to infect the white rat and guinea pig with the A$_{48}$ and B$_{51}$ strains although complement fixing antibodies were produced in the guinea pig The ferret and nutria also were found to be refractory to strain A$_{48}$ Madin and Traum (1953) reported lesions in the guinea pig using field material from outbreaks in 1936 and 1949 In the 1936 outbreak, passage was made in 2 animals in 1949, 5 animal passages were made In both instances, however, the virus was not viable when tested in swine Brooksby (1954) reported negative results with strains 1934 B and 1943 101 in suckling and young adult white mice Bankowski and Wood (1953) found that dogs were irregularly susceptible to types A$_{48}$ B$_{51}$, and C$_{52}$ Intradermal lingual injection produced mild lesions at the points of inoculation, characterized by erosion of the epithelium blanching, and extension The virus was recovered from the spleen of one febrile but not from 2 afebrile dogs

The limited host range prompted investigations in the field of tissue culture McClain et al (1951) reported the first successful cultivation of VESV, demonstrating that strain B$_{51}$ could be propagated on embryonic swine skin and that cytopathogenic effects were produced Subsequently Madin et al (1958a) propagated the virus on monolayer cultures of adult swine kidney, testicle, lung, and amnion as well as canine feline, and equine kidney Bankowski and Pfeiffer (1955) have propagated the B$_{51}$ strain in a medium composed of Baker s fluid and minced swine embryos, harvested from sows in the third to fifth week of gestation In addition, Bankowski (1954) states that this virus also can be cultivated on embryonic swine skin transplanted to the chorio allantoic membrane of chick embryos

ECONOMIC IMPORTANCE

Vesicular exanthema is of great economic importance, since it causes serious weight losses in fat hogs, slow gains in feeder stock, deaths in suckling pigs, abortions in pregnant sows, and impaired lactation in nursing sows In addition, it is clinically indistinguishable from foot and mouth disease (FMD) and vesicular stomatitis (VS) in swine, thus requiring expensive quarantine procedures

The cost of a debilitating disease such as VES is always difficult to assess The annual loss during the period 1912-51 was estimated at $887,000, during which time the disease was confined to California

FIG 10 2—Size and weight comparison of two 5 month old litter mates The larger animal (73 lb) was protected against ves cular exanthema by the inoculation of immune serum the smaller (28 lb) acting as an untreated serum control Note difference in size and weight due principally to the effects of the virus

which time the disease was confined to California (Duckworth, 1953a)

In 1948 and again in 1949, the virus appeared in swine en route to the port of Honolulu These animals had been loaded from California ports and had apparently come in contact with the virus prior to or during shipment Prompt quarantine and slaughter before reaching Hawaii *prevented the spread of the disease to the Hawaiian mainland*

On June 16, 1952, VES appeared in Grand Island, Nebraska, at a plant manufacturing biologicals The source of the infection was traced to Cheyenne, Wyoming, where hogs had been fed garbage from transcontinental trains whose point of origin was California It is assumed that contaminated pork scraps were the source of the virus Before the disease was detected in the herd at Grand Island, some of the hogs had been shipped to the Omaha stockyards and resold In this manner, the disease spread rapidly and by July 29, just 43 days after discovery of the disease in Nebraska, 19 states were placed under federal quarantine for VES On August 1, 1952, a state of emergency was declared by the Secretary of Agriculture, thus providing federal support for an active eradication program including slaughter and payment of indemnities (Simms, 1953)
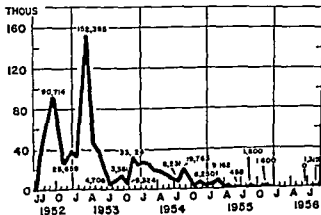
FIG 10 1—Numbers of vesicular exanthema infected swine from June, 1952, to September, 1956 Figures for California are *not* included
(Courtesy U S Dept of Agriculture)

From June, 1952, to September, 1953, a total of 42 states and the District of Columbia *had experienced the disease Figure 10 1 shows the numbers of infected swine* from the period, June 2, 1952, through July, 1956

Figures for the state of California are not included in these totals due to establishment of the disease in the raw garbage-feeding areas of this state *The number of outbreaks has steadily declined throughout the nation, so that only 3 outbreaks, all in New Jersey, were recorded in 1956* California had 15 outbreaks in 1955 (Myers 1955), and since October of that year no further outbreaks have been recorded

HOST RANGE

Vesicular exanthema of swine virus (VESV) shows a marked specificity for porcines and an almost equal indisposition for other species

Traum (1933), the first to study the host range of the virus, found in the original outbreak of 1932 that inoculation of material into guinea pigs, swine, and a limited number of cattle and horses produced lesions only in swine In the 1933 outbreak, inoculation of the same species provided consistent "takes" only in swine, with mild reactions in 4 of 9 horses These findings were confirmed by Reppin and Pyl *(1935), and Mohler (1933a), who found* the horse to be easier to infect than previously suspected Crawford (1937) identified strains of the virus, A, B, C, and D, and found that while all four were infective for swine, only types A and C were infective for the horse Crawford (1937) attempted passage of the virus to sheep goats, guinea pigs, white rats, white mice and hedgehogs, and found that none of the 4 strains produced any visible reaction in these species British workers (British Report, 1937), using an unspecified strain infected swine, but not horses, cattle sheep, goats, guinea pigs, rats *(Rattus norvegicus)*, or hedgehogs. Madin and Traum (1953) reported negative results with the chick embryo, rabbit, and several strains of adult and suckling mice including

2 Sal soda used at the rate of 13½ oz to 1 gal water

3 Lye (sodium hydroxide) used at the rate of 13 oz to 5 gal water

The viricidal effect of these compounds apparently depends on a very high pH which denatures the virus protein Such compounds have also found acceptance as viricidal agents in FMD (see Chapter 12) and against VS (Olitsky *et al*, 1928)

Antigenic Types

The existence of a plurality of virus types was demonstrated by Crawford (1937) with virus material collected from the outbreaks in 1933 and 1934 Four immunological types were found in swine based on cross immunity tests These were named A, B, C, and D Types B and D were infectious for swine only, whereas A and C were infectious for both horses and swine Types B and D caused more severe reactions in swine than either A or C In 1940–42, three immunologically distinct types were recovered in California but were subsequently lost. Two of the earlier types are still available, the 1934 B and the 1943 101 strain collected by Traum (Brooksby, 1954) All other strains collected prior to 1948, however, are not available In December, 1948, Madin and Traum (1953) isolated A_{48} In 1952, Bankowski *et al* (1953) reported the isolation of strains B_{51} and C_{52}, and in 1953 strain D_{53} (Bankowski *et al*, 1954) Brooksby (1954) has compared the first 5 of these strains by complement fixation and has found them to be distinct antigenic types

In July of 1954, Bankowski *et al* (1955) isolated still another immunological type, E_{54} In this same publication, attention was directed toward 2 potentially new types then under trial, which Bankowski *et al* (1956) have subsequently called F_{55} and G_{55} The last named 2 strains and a third isolate, as yet unidentified, were all characterized by their low pathogenicity for swine At the present time there are at least 7 known immunological types, with

the possibility of 9 — if those in the possession of the British workers are distinct from the 7 reported in this country It is possible that as long as VESV finds susceptible hosts wherein to multiply, the number of types may continue to increase

Complement fixation and serum neutralization tests corroborate the immunological identity of the types Bankowski *et al* (1953) have demonstrated that the types can be separated by complement fixation in spite of some cross reactivity Brooksby (1954) has confirmed these results Mc Clain *et al* (1954) separated the A_{48} and B_{51} types by complement fixation and *in vitro* neutralization The use of the serum neutralization technique is discussed in the section on diagnosis (page 181)

Infectivity

Following intradermal inoculation of swine with VESV, Madin and Traum (1953) found that the virus could be recovered from the blood at 24 and 48 hours, but not at 72 or 96 hours, after inoculation In a similar experiment, Patterson and Songer (1954) found blood samples positive only at 48 and 72 hours after inoculation These workers found, however, that if the donor' animal were inoculated intravenously, the period of viremia was 72 to 84 hours, beginning about 48 hours prior to vesiculation in the donor' pigs and ending 36 hours after vesiculation Mott *et al* (1953) slaughtered a group of inoculated swine approximately 6 hours prior to the development of vesicles (30 hours after inoculation) All susceptible swine fed feet, snout, spleen crushed bone, whole blood, lymph node viscera and muscle, developed clinical VES Animals fed feces and urine failed to develop a clinical infection but were immune to subsequent challenge, indicating the presence of virus in quantity sufficient to stimulate immunity It appears then that the virus quickly becomes widespread throughout the hog s body

Patterson and Songer (1954) extended these experiments by inoculating each of 12 swine intravenously and then sacrificing

(Agricultural Research Service Report, 1954). The cost to the United States from 1952 to 1955 was estimated at $33,000,000. This figure included losses to the swine industry of $17,000,000, to hog cholera virus serum producers of $5,000,000, and to state and federal control programs of $11,000,000 (Mulhern, 1956).

DISTRIBUTION

The naturally occurring disease has been reported only within the United States (Madin and Traum, 1955).[1]

ETIOLOGY

VES is caused by a filtrable virus, VESV.

Physical-Chemical Properties

The inability to obtain significant quantities of virus material from swine, and its restricted host range have materially retarded progress on the physical and chemical characterization of this agent. The successful cultivation of the virus in tissue culture, however, may provide the necessary stimulus and the means whereby the virus can be more fully characterized (Madin et al., 1958a).

Size

The size of the virus was calculated to be from 13 to 20 mμ, since it was capable of passing gradacol membranes of 44 mμ average pore diameter (APD), but not 34 mμ APD (Madin and Traum, 1953). Brooksby (1954) reported that the 1934 B and 1943 I01 strains passed gradacol membranes of 110 mμ and 70 mμ APD but not 37 mμ APD.

Resistance

The virus has been preserved for as long as 2½ years at ordinary refrigerator temperatures in the form of unground vesicle coverings stored in 50 per cent glycerin-phosphate buffer. It will retain its infectivity for as long as 6 weeks at room temperature in one per cent ordinary peptone solution and will survive for at least 24 hours at 37° C. in Sorensen's buffer. Storage at −27° C. is routinely used. Mott and Patterson (1956) report that VESV is inactivated at 62° C. for 60 minutes and 64° C. for 30 minutes. It is not inactivated at 64° C. for 15 minutes, 60° C. or 62° C. for 30 minutes, nor 60° C. for 60 minutes. (See also Mott, 1956.)

In a series of feeding experiments, Mott et al. (1953) showed that contaminated meat scraps were infectious after storage at 7° C. for 4 weeks and at −70° C. for 18 weeks. Traum and White (1941) placed infected vesicle coverings inside the bone marrow cavity of both cured and fresh hams, refrigerated them overnight, then "cooked" them at 184° F. under 10 lb steam pressure for 10 minutes in a garbage cooker without completely destroying the infectivity of the vesicle coverings. Mulhern and Patterson (1956), however, claim that the virus is inactivated in hams cooked at 150° F. for 30 minutes. In certain cases where viral suspensions had lost their infectivity, Madin and Traum (1953) found it possible to "reactivate" them by the addition of cysteine monohydrochloride to the viral suspension. The minimum period necessary to "reactivate" was found to be 8 days, and once "reactivated," the infectivity was retained for 262 days, the longest period tested.

Data regarding the action of viricidal agents are not available. The use of sodium hydroxide in the form of readily available 2 per cent lye solution has been recommended by Madin and Traum (1953), and Mott et al. (1953). Mulhern and Patterson (1956) stated that a 2 per cent lye solution kills the virus. The Bureau of Animal Industry Order No. 383, Rev. (1953) requires the use of one of the following disinfectants:

1. Soda ash (sodium carbonate) used at the rate of 1 lb. to 3 gal. water.

A_{48}, C_{62}, D_{53}, and E_{64} multiply and pro duce CPC in canine kidney The remain ing types B_{51}, F_{55}, and G_{55} do not pro duce consistent CPC in this tissue host, but the F_{55} type appears to be synthesized even in the absence of CPC Strain A_{48} multi plies and produces CPC irregularly in equine kidney CPC are apparently not produced on monolayer kidney cell cul tures of sheep, cow, monkey, guinea pig, rabbit, mouse, hamster, rat, goat, or on HeLa, L cells, or chick fibroblasts CPC are produced by all types on first passage in feline kidney but usually are not visible after the second passage It has not yet been determined whether or not virus is being synthesized in the absence of CPC as is the case with type F_{55} in dog kidney VESV grown in monolayers of swine kid ney has a cytopathogenic endpoint between 10^7 and 10^9 TCID$_{50}$ units per ml , while the same viral suspension titrated in swine has an ID$_{50}$ endpoint of between 10^4 and 10^6 infective particles per ml

CLINICAL SIGNS

The introduction of virus into suscep tible swine usually produces a characteristic rise in temperature followed by vesicles at one or more of the following sites snout, lips, tongue, and mucosae of the oral cavity, and on the sole, interdigital space, and coronary band of the foot Occasionally lesions may appear on the teats particu

larly of nursing sows (Hurt, 1940–41, Traum, 1936) and on the skin covering the metacarpus and metatarsus (British Re port, 1937) Inoculation of the virus in tradermally into the snout or mucosae of the oral cavity, or both, by needle or scari fication usually produces the classical re action first the "primary" lesions appear at the site of inoculation within 12 to 48 hours, and then 'secondary' lesions de velop elsewhere 48 to 72 hours later Inoc ulation of the virus via the subcutaneous, intramuscular, or intravenous routes is usually followed by the appearance of vesicles at any of the susceptible sites with in 24 to 96 hours

In the typical case, a diphasic sympto matic response results Phase 1, lasting from 48 to 72 hours, is marked by pyrexia and the appearance of primary vesicles, usually associated with anorexia and list lessness The primary vesicles consist of blanched, raised areas of epithelium vary ing from 5 to 30 mm in diameter, and from 10 to 20 mm in height, and filled with a serous fluid rich in virus Such vesicles resemble the blister' formation accompanying burns or excessive dermal friction The epithelial coverings may ' lift' with the slightest pressure, revealing a raw, bleeding, and exceedingly sensitive corium which is subsequently covered by a yellowish fibrinous membrane (British Report, 1937, Traum, 1934, Crawford, 1937)
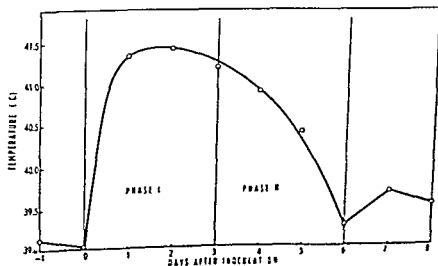
FIG 10 5–Characteristic temperature curve follow ing intradermal inocula tion of swine with vesicular exanthema virus (Madin and Traum, 1955)

the animals in pairs at 1, 2, 5, 7, 14, and 30 days after inoculation. Meat, lymph node, heart, lung, spleen, liver, kidney, blood, and crushed bone, pooled from each pair of donor pigs, was fed to different groups of 5 susceptible swine. The material obtained from the animals slaughtered at 1, 2, and 5 days proved to be infectious; the material taken at 7 days did not produce clinical VES but did produce immunity, and the material taken at 11 and 30 days produced neither infection nor immunity. These studies show that the virus remains viable in certain of the tissues of the infected animal for at least 7 days.

The ID_{50} of fresh vesicle covering material has been shown by Mott et al (1953) to be $1 \times 10^{-5.3}$, which is in close agreement with the figure of 1×10^{-6} suggested by Madin and Traum (1953). Comparative titrations using infected vesicle covering material or infected, defibrinated blood indicated that it takes 10 to 100 intradermal snout minimal infecting dose (MID) to make one MID via the oral route.

Behavior in Tissue Culture

The demonstration by McClain et al (1951), that VESV grew and produced cytopathogenic effects in tissue culture of swine origin, opened the possibilities of expanded research with this virus since, for the first time, an experimental host other than swine was made available. In addition, the method of plaque assay described by Dulbecco (1952) could be used to assay and study VESV, an observation subsequently substantiated by Sellers (1955). Recent observations have shown a remarkable variation in plaque morphology among the seven antigenic types. In addition to inter type variation, intra type variation has been observed characterized by the presence of large, clear plaques and minute, opaque plaques. The minute plaques exceed the large plaques in number, the ratio varying from approximately 1:2–3 with A_{48} to more than 1:100 with B_{51} and E_{54}. Clones of the large and minute plaque variants have been produced by

FIG. 10 3—Cytopathogenic changes produced by vesicular exanthema virus on monolayer of swine kidney tissue culture, 48 hours after inoculation.

techniques of plaque purification and it has been shown that the minute plaque former is essentially avirulent in swine, while the large plaque former is virulent. The virus found in different plaques appears antigenically indistinguishable despite the marked differences in virulence (McClain et al, 1958).

The present status of our knowledge indicates that all antigenic types will multiply and do produce cytopathogenic changes (CPC) in monolayers of swine kidney, lung, liver, testicle, and amnion. Types

FIG. 10 4—Vesicular exanthema virus plaques produced on monolayer of swine kidney cells 48 hours after inoculation

The primary lesions on the snout usually
spread to and involve the adjacent mucosae
of the lips, cheeks, and tongue This spread
is probably caused by virus liberated from
the primary vesicles, since new lesions often
follow the path taken by fluid escaping
from ruptured vesicles The subcutaneous
tissues of the snout and tongue may be
come hyperemic, swollen, and sensitive to
pressure As a result, the snout may appear
bulbous, and the swelling of the tongue
lead to slobbering (Hurt, 1910–41)
Phase 1 is almost invariably accompanied
by an increase in body temperature to 105
or 106° F Occasionally 108° F is reached
The end of phase 1 is usually signified
by a decline in temperature to below
101° F and rupture of the "primary" vesi
cles

Phase 2 is ushered in by the formation of
'secondary' vesicles on the soles of the
feet, between the interdigital spaces and at
the junction of the epithelium and nail of
the foot (coronary band) The appear
ance of foot lesions is usually indicated by
a characteristically hesitant gait, commonly
described as "ouchy" The animal may
continue to walk in this halting fashion or
may refuse to move until the pain and
swelling have decreased In severe attacks,
an edematous swelling of the legs and
joints may be present

Phase 2 usually lasts for 24 to 72 hours
when the secondary vesicles rupture the
pain subsides, and normal living habits
are resumed During both phases 1 and 2

FIG 10 9—Freshly ruptured vesicles on the sole
and coronary band of the hind feet Note the
hyperemic corium

the animal may refuse food and this
coupled with the severe pyrexia literally
melts the fat from market animals

Recovery of uncomplicated cases is
usually prompt and without sequelae The
healing of very severe foot lesions may
result in the formation of nodules of granu
lation tissue which arise from the sole of
the foot prior to replacement by the

FIG 10 10—Chronic foot
lesion showing replacement
of epithelium with granula
tion tissue (Courtesy
Dr E. R. Quortrup.)

FIG. 10.6—Tongue lesion showing ruptured vesicles, 72 hours after inoculation.

FIG. 10.7—Unruptured vesicle involving sole and coronary band of lateral digit of right hind foot.

FIG. 10.8—Typical snout lesions shortly after rupture of vesicles.

DIAGNOSIS

The diagnosis of a frank vesicular disease is not difficult since the clinical signs of pyrexia, vesiculation, and lameness are almost invariably present

The presence of occult infections as reported by Mott et al (1953) and by Ban kowski et al (1955) are difficult, however, if not impossible, to determine by simple clinical methods   The similarity of the clinical syndrome produced by VESV, VSV and FMDV in swine makes the differential diagnosis of a vesicular disease difficult   The present method of differentiating between VES, VS, and FMD depends on the differential susceptibility of various test animals   This system is discussed in detail by Traum, Callis, and Shahan in Chapter 12

The weakness of this method of diagnosis has been pointed out by Madin and Traum (1953)   This system of animal inoculation is satisfactory as long as live virus is available, speed is not critical, typing of the individual virus is not required and a new vesicular disease has not arisen This weakness was illustrated clearly in the initial outbreaks of VES in 1932 and 1933, when a positive diagnosis could not be reached   Because of the drawbacks to the animal inoculation system, the investigation of serological methods has received attention   Bankowski et al (1953) announced the development of a complement fixation test capable of identifying and differentiating the antigenic types of VESV Brooksby (1954) has modified their technique by the addition of sodium poly anetholesulphonate to the swine complement for the purpose of destroying the third component (C₃)   These initial complement fixation techniques have improved diagnosis in field outbreaks   In addition, Bankowski and Kummer (1955) have been able to distinguish VESV strains A₄₈, B₅₁, C₅₆ and D₅₃ from VSV by their test.

McClain et al (1951) briefly described a method by which a serum neutralization test could be done in tissue culture by taking advantage of the fact that cyto

pathogenic effects were not seen in the presence of specific immune sera   Madin et al (1958b) have extended these studies and have shown that various strains of VESV could be separated and VESV differentiated from VSV by this method

The test consists of mixing known amounts of virus with suitable dilutions of homologous and heterologous hyper immune rabbit antisera   The virus serum mixtures are incubated at room temperature for 90 minutes following which 1 ml amounts are dispensed into tubes of swine kidney tissue culture, 8 tubes per dilution Control tubes of virus dilutions, mixed in equal volumes with the diluent and sub jected to identical treatment as the virus serum mixtures, are always included   All tubes are incubated at 37° C for 72 hours Titration results are based on the presence or absence of observable cytopathogenic changes   The results of a typical neutralization test using 7 strains of VESV against a single dilution of antisera are shown in Table 10 2   It can be seen from these results that large amounts of virus are neutralized by the homologous serum while the heterologous serum produces practically no detectable neutralization

This technique makes possible the rapid and accurate differentiation of the strains tested through 1957

Hemagglutination has been tried repeatedly by Madin and Traum (1953) with strain 101, and by Crandell and Madin (1957) with strains A₄₈ and B₅₁ with sheep, rabbit, guinea pig, ferret, hamster, rat, swine, and human type O erythrocytes, all with negative results

The problem of diagnosis should not be left without a word concerning the isolation of VESV from field outbreaks   The current method of obtaining viral material is to remove the epithelial vesicle coverings from the snout or feet of infected swine (preferably those with temperatures of 105° F or higher) and to preserve these epithelial coverings in 50 per cent glycerin phosphate buffer pH 7 1 at 34 to 40° F (Madin and Traum, 1953)   Inocula can

normal epithelium. Pyogenic bacteria may gain entrance through the damaged epithelium and may cause severe or fatal secondary infections. In a certain proportion of cases, the hoofs are lost from the infected feet and replacement may take from 1 to 3 months, during which time the animal may be partially lame. The junction of the old and new nail is then marked by a dark brown or black line, making a diagnosis of VES infection probable even though all acute symptoms have disappeared.

In addition to the above described signs, Hurt (1940–41) has called attention to the severe attacks of diarrhea occasionally accompanying the infection, to an apparent increase in the abortion rate of infected sows, and to a general drop in milk production in lactating sows. Wicktor and Coale (1938), and Mott *et al.* (1953) warn that a mild infection may be missed completely, thus supplying a source of "occult cases."

PATHOLOGICAL CHANGES

Vesicle formation is the only known lesion directly attributable to the infection. The virus appears to multiply principally in the Malpighian layer of the epidermis. In the course of this process, the individual stratified squamous epithelial cells undergo marked swelling of the cytoplasm, eventually producing ballooning degeneration. These cytoplasmic changes are usually accompanied by pyknosis and karyorrhexis of the nuclei.

As the cells become necrotic in a localized area, the virus spreads from cell to cell, thus involving large numbers of cells in a given circumscribed area. This process is repeated in different areas throughout the epithelial sheet The necrosis and subsequent dissolution of the virus infected cells leaves the epithelial sheet perforated by a series of "holes" surrounded by intact epithelial cells. Those cells at the edges of the lesions usually show early evidence of degeneration accompanied by a marked stretching of the

intercellular bridges and considerable intercellular edema.

The subcutaneous tissues show congestion, edema, and, occasionally, hemorrhage. Polymorphonuclear leukocytes are present in large numbers throughout the dermis and infiltrate into the lesions in the Malpighian layer. It is not known whether this acute inflammatory response is due directly to the multiplication of virus or is a response to the death of the epithelial cells.

The progressive weakening of the Malpighian layer accompanying virus multiplication in this area, coupled with the increasing pressure of the edema fluid, forces the intact upper layers of the epidermis above the surface of the noninfected skin, thus producing the characteristic raised vesicle. Inclusion bodies have not been reported.

The pathologic changes of VES in the skin are very similar to those described by Chow *et al.* (1951) for VS and by Galloway and Nicolau (1928) for FMD.

FIG. 10.11—Histology of typical vesicle formation. Note the infiltration of acute inflammatory reactive cells in stratified squamous epithelium. (Madin and Traum, 1955.)

a method of producing antigenic material in quantity The current studies by Madin *et al* (1958b) show that any desired quantity of antigenic material can be produced on swine kidney monolayer cultures Bankowski and Pfeiffer (1955) reported that VESV could be propagated in quantity using suspended cultures of minced swine embryos Although the technical problems of producing a vaccine appear to have been solved, no vaccine has appeared There are 3 possible reasons for this situation (1) The urgency for such a product has diminished, due to the current success in controlling the disease by feeding cooked rather than raw garbage to swine and by enforcing quarantine measures (2) The continuing multiplicity of new antigenic types of VESV suggest a vaccination problem similar to that encountered in FMD (3) The research facilities required for large scale vaccine trials are not readily available

EPIZOOTIOLOGY

VES is known to be spread by at least 2 methods, namely, direct contact and the feeding of raw garbage These 2 routes of infection can account for the vast majority of the outbreaks, excepting the initial outbreaks of 1932 and 1933, and the subsequent epizootic of 1934

Direct contact includes, for purposes of this discussion, contact with contaminated feed, water, and fomites, as well as contact with infected animals within the hog's particular environment It should be pointed out that, as a group, swine live in most intimate contact, and the exchange of disease agents can occur constantly by either immediate or mediate contact It may be for this reason that VES shows no particular seasonal incidence inasmuch as the environment suitable to it is reasonably constant.

The work of Mott *et al* (1953) is of particular interest in the matter of direct and indirect contact infection They placed groups of susceptible swine in direct contact with infected donor animals which had been inoculated 12, 24, 36, 38, 72, 96, 120, 144, 192, 240, and 288 hours prior

to the contact They found that the susceptible animals contracted the disease from those inoculated during the intervals from 12 to 120 hours but not from those inoculated prior to that time The authors suggest that the donor animals did not excrete virus in infective quantities later than 120 hours after inoculation In another series of experiments, 2 infected animals were placed in contact with 2 normal swine in a clean pen After 12 hours of contact, the 2 donors were withdrawn and placed in a second pen with 2 other normal hogs The donor hogs were moved from pen to pen at intervals of 24, 36, 72, 96, 144, and 192 hours after they were inoculated In each recipient pen, one of the normal animals was scarified on the snout and feet prior to the introduction of the infected donors Both donor animals showed clinical VES 48 hours after inoculation The normal animals showed clinical VES in those groups exposed during the intervals of 24, 36, 48, 72, and 96 hours, but not in the intervals of 12, 144 and 192 hours These data indicated that virus was not eliminated by the donor animals prior to 24 hours but began shortly there after and continued until 96 hours after inoculation The extent of environmental exposure by infected swine was illustrated by the following experiment Two normal contacts were placed in each of 8 infected pens at 0, 24, 48, 72, 96, 120, 144, and 168 hours after removal of infected swine Only one of the normal contacts developed lesions and this animal belonged to the 72 hour group Subsequently, it was shown by challenge with live virus that both animals in the 72 hour group and one in the 0 hour group had been infected with the virus This pattern of indirect exposure was similar to that found by Crawford (1937)

What then is the role of raw garbage as a vehicle of spread? According to Duckworth (1953a), "Raw garbage is the source of VE ' Support for his statement can be found in Table 10 1, which shows the correlation between raw garbage feeding and outbreaks of VES in California Mulhern (1953) has reported that almost

TABLE 10 2

RESULTS OF NEUTRALIZATION TESTS USING 7 STRAINS OF VESV AGAINST HETEROLOGOUS* AND HOMOLOGOUS† RABBIT ANTISERA IN SWINE KIDNEY TISSUE CULTURE SYSTEM

VESV Type	TCID50‡ Virus Titer	Logs Virus Neutralized						
		Type Specific Antisera (Rabbit)						
		A	B	C	D	E	F	G
A_{48}	7 4	6 0§	0	0	0 6	0	0 8	0 5
B_{51}	7 0	0 1	5 2	0 1	1 0	0	0 5	0 5
C_{52}	7 4	0	0 2	6 4	0 1	0 5	0 5	0 1
D_{53}	6 5	0	0 1	0	4 4	0 5	0	0 3
E_{54}	7 2	0 3	0 6	0	4 4	4 0	0	0
F_{55}	6 3	1 0	0 3	0 2	0	0 5	5 1	0 5
G_{56}	5 5	0	0	0 2	0 2	0	0	4 0

* Heterologous antisera diluted 1 10
† Homologous antisera diluted 1 100
‡ TCID50 = tissue culture infecting dose capable of destroying 50 per cent of swine kidney tissue cultures
§ Figures in body of test are actual, based on several replicate series Logs of virus neutralized of 1 0 or less are not considered significant

then be prepared by grinding the epithelium and suspending it in an appropriate diluent

An additional method is now available Small portions of vesicle covering material may be taken directly from the hog and placed in tubes of swine kidney tissue culture After incubation for 24–48 hours, the presence of cytopathogenic changes indicates the presence of a cytopathogenic agent which may be accurately identified by serum neutralization These methods of virus isolation and identification for tissue culture techniques should simplify and improve the speed and accuracy by which not only VESV, but FMDV and VSV, may be routinely isolated, typed and investigated (Madin et al, 1958b)

TREATMENT

No treatment for VES is known Certain precautions of a palliative nature may be taken, which will tend to reduce the economic losses from the infection Weight losses can be reduced if infected animals are fed only soft feeds or slops, if they are taken off concrete or similar hard surfaces, and if adequate amounts of clean water are kept before them at all times Where infected animals must be maintained in crowded quarters during rail shipment, in feed lots or in slaughter houses, secondary bacterial complications may be controlled by the judicious administration of antibiotics (Madin and Traum 1955)

IMMUNITY

Animals which have recovered from a clinical attack of the disease are solidly immune for at least 6 months to reinfection with the same antigenic type (Madin and Traum, 1953) Mott (1956) reports that immunity persists for at least 20 months in 50 per cent of animals convalescent to type B_{51} The presence of neutralizing antibodies may be detected as early as 10 to 12 days after infection and continue to climb to a peak between 21 and 28 days post inoculation (Madin et al, 1958b)

Passive immunity may be conferred against types A_{48} and B_{51} for a period of 11 to 21 days by the inoculation of type specific immune sera (Madin, 1951, 1956b) Active immunization by means of vaccines has found no application to date Preliminary trials with a formalinized vaccine made from infected epithelial coverings protected swine against direct intradermal challenge of the homologous strain for at least 6 months (Madin and Traum 1953) These same authors pointed out that the production of a usable vaccine must await

derstanding of the epizootiology of the disease, no outbreaks were reported during the 42 month period between June 20, 1936 and December 4, 1939. During this time all of the swine practices had been continued as usual, and contrary to the situation which prevailed in 1932, 1933, and 1934, there was contaminated pork in circulation in the trade channels—since from 1934 to 1936, a total of 127,000 infected animals had gone to slaughterhouses in the state Thus, while all the known means of transmitting the disease were at hand, no outbreaks were reported. Currently then, we have no satisfactory explanation as to the actual source of the virus in 1932-34

From the earliest outbreaks until the present time, it has been noted that the percentage of animals infected on any given premises or within any given group varies considerably with the outbreak in question (Duckworth, 1953a, Hurt, 1940-41) Recently, Bankowski et al (1955) studied the morbidity rates in natural outbreaks caused by 4 antigenic VESV types, namely, B_{51}, C_{52}, D_{53}, and E_{54} They found that the rates varied from less than 10 per cent to as great as 100 per cent within a given group, and that there was no consistent correlation between strains and morbidity Furthermore, the clinical severity of the disease was not related to morbidity Currently, neither the attack rate nor the severity of the disease can be predicted or assessed in terms of any known factors The earlier observations of Crawford (1937), that severity of the clinical symptoms was directly related to the antigenic type of the virus, may have been true for particular field conditions or may have been due to the fact that these virus types behaved differently from those available today

The role of the various antigenic types in the epizootiology of the disease is not clearly understood The ease with which different antigenic types have been isolated indicates that the virus is extremely flexible in its basic antigenic structure A study of the various antigenic types present

in California and in other states from October, 1951, to June, 1955, was made by Bankowski et al (1955) During this period a total of 325 field samples was received, 126 of these originating from outbreaks native to California, the remaining 199 representing materials obtained from swine brought into California From the latter group, 139 were typed and all were found to be B_{51} From the California samples, 88 were studied and these showed the following type distributions A_{48}, 0 per cent, B_{51}, 43 per cent, C_{52}, 27 per cent, D_{53}, 23 per cent, and E_{54}, 7 per cent. These same authors have further shown that one immunological type tends to be predominant and is replaced by another in fairly rapid sequence Thus, in 1951, type B_{51} was predominant in California this was replaced in 1952 by type C_{52}, in 1953, type B_{51} reappeared, and early in 1954, type C_{52} reappeared From May, 1954, through June, 1955, type D_{53} appeared to predominate, although type E_{54} was present in some of the outbreaks during this same time period Mott and Patterson (1956) state that the outbreak of VES in the state of New Jersey in the fall of 1956 was caused by an unknown virus type, which was not type A_{51}, B_{51}, or C_{52} (see also Mott, 1956)

The ability of the virus to appear as different antigenic types means that active immunity to one virus type does not necessarily remove such animals from the reservoir of susceptibles In fact, the immune animal may more accurately represent the environment within which subsequent mutation of the virus to a different antigenic type is fostered This latter point has been illustrated by Bankowski et al (1955) in a study of recurrent infections in a single herd of swine Two distinct outbreaks of the disease were found in this herd within a period of 40 days, each outbreak being caused by a different virus type

It would appear then that we are faced in the case of VES with some of the same general epizootiological problems that have vexed us so long in FMD, namely, the oc

all of the outbreaks occurring after the 1952 "escape" of the virus from California have either had direct or indirect connection with garbage feeding establishments The link between raw garbage and the virus apparently is infected raw pork scraps which serve to transmit the disease to susceptible swine This hypothesis gains theoretical support from the feeding experiments conducted by Mott *et al* (1953), by Patterson and Songer (1954), and from the unpublished studies on the survival of the virus by Traum and White (1941) These experiments demonstrate that the virus can survive in an infected carcass and eventually find its way back to susceptible swine through raw garbage Indirect evidence for this assumption is provided by the fact that cleanup and disinfection did not prevent the almost constant recurrence of the disease on raw garbage feeding ranches in California

Whereas this mode of spread explains many of the outbreaks, it seems inadequate, for example, for the 1932, 1933, and 1934 outbreaks Prior to 1932, no vesicular disease other than FMD had been reported in swine It is particularly significant to recall that California had experienced FMD in 1924, 1925, and again in 1929, as a result of which all regulatory officials were *particularly alert to a vesicular disease outbreak* We can be reasonably certain that the 1932 outbreak of VES was *the first to occur and had its origin in one of the areas described in the section on History* From the evidence available at that time and from a subsequent review of this evidence, the outbreak in 2 of the areas (Orange and Los Angeles counties) was not related to the outbreak in San Bernardino County Thus, 2 separate foci were apparently present almost simultaneously The ranches in Orange and Los Angeles counties obtained their garbage by contract from domestic source only The San Bernardino County premises could possibly have had garbage purchased from a foreign ship through a contract with the city of Long Beach, but it is highly doubtful that any significant amount of such

garbage found its way to the hog ranches In this respect, it is important to remember that, since the 1929 outbreak of FMD in California, a regulation had been in effect forbidding all ships to bring garbage into American ports

In 1933 the second outbreak occurred this time 100 miles south of the 1932 occurrence, but again on a garbage feeding hog ranch The only known association between the 1932 and 1933 outbreaks, outside of raw garbage, was that 2 of the ranches involved — one in the 1932 and the other in the 1933 outbreak — were both operated by the same owner There is no evidence, however, that man had been instrumental in transmitting the disease in 1933 As nearly as could be ascertained the 1933 outbreak was a distinct and separate outbreak, similar to the 1932 occurrence In 1934 the third outbreak occurred, again on a garbage feeding hog ranch 500 miles distant from the 1932-33 foci In discussing the 1934 outbreak Duckworth (1953a) pointed out that It is inconceivable that infective material of any kind could have carried over from either of the two earlier outbreaks and *found its way 15 to 26 months later into a swine herd 500 miles distant* In addition it should be remembered that all animals in the 1932–33 epizootic were slaughtered and buried, and, therefore, these infected carcasses did not enter into the normal trade channels and could not have contaminated raw garbage with virus Thus it appears that the 1934 outbreak represented still another separate and distinct focus

The source of the virus in the first 3 outbreaks is difficult to explain Shope (1955) has suggested that VES may be primarily a disease of some wild animal and that domestic swine happen to be mutually susceptible Hog ranches which feed raw garbage may attract such a reservoir and in the course of events, swine may be brought into suitable contact with the infection No experimental evidence is at hand to support, or exclude, such a hypothesis To further complicate an un

Bureau of Animal Industry Order No 383 of June 20, 1953, which in part established that swine that had been fed raw garbage at any time could not be shipped inter state except for special processing The net effect of this regulation was to reduce the actual market value of such animals, thus placing an economic penalty on the prac tice of feeding raw garbage Initially, this regulation was not rigidly enforced, in order to give hog raisers time in which to set up facilities for cooking garbage, and in order that adequate personnel could be obtained for routine inspection of the gar bage cooking facilities In lieu of this pro cedure, swine which had not been fed raw garbage for the preceding 30 days or had not been in contact with swine fed raw garbage for the preceding 30 days, were allowed to be shipped interstate without processing precautions

As equipment and inspection personnel became available, the regulation became fully effective January 1, 1956 On or after that date, swine raisers who continued to feed raw garbage had or will have three alternatives

1 Begin the cooking of garbage

2 Conform to restricted marketing in some instances within the state

3 Conform to restricted marketing in terstate for processing only

The results of these so called "garbage cooking laws' have been extremely en couraging As of August 31, 1956, only 9 4 per cent of garbage fed swine were still being fed raw garbage, this figure repre senting less than 0 4 per cent of all swine produced in this country The problems, from then on, center around adequate inspection of garbage cooking operations and toward continued and vigilant oppo sition to any tendency which detracts from the efficiency of garbage cooking regula tions Those interested in the technical details of processing raw garbage so that it may safely be used as swine feed should consult the papers of Long and Johnson (1952, 1953) In addition, technical and economic data are to be found in the Special Report of the Joint Legislative Committee on Agriculture and Livestock Problems from the state of California (1955)

The effect of the control measures in dicated herein has been extremely en couraging On the national scene, the inci dence of new outbreaks has dropped from a high of 777, between November, 1952, and April, 1953 to 16 in 1955, and 3 in 1956 In California, state quarantine regu lations permitting the Director of Agricul ture complete control of garbage-fed swine were put into effect in March, 1954 These included restricting the movements of swine or pork products produced on raw garbage, the liquidation of the remaining infected swine, and the conversion from raw garbage to cooked garbage, all within a 12 month period These combined efforts effectuated a marked decline in the num ber of VES outbreaks In less than one year 97 per cent of the raw garbage feeding premises had converted to cooked garbage As the number of raw garbage feeding ranches declined, so did the outbreaks of VES For example, in 1953, there were 137 outbreaks recorded, in 1954, only 19 This declined to 15 in 1955, and since Novem ber, 1955, no outbreaks of VES have been reported in California

The role of prophylactic agents in the control of the disease has not been eluci dated Madin (1954, 1956b) has suggested the use of immune sera to confer tempo rary immunity in situations where such would be of value Active immunization as already mentioned needs considerable re search before its role can be evaluated

At present, a considerable measure of success has been achieved by interrupting the known infection chains The possibility still exists however, that this virus has other means of being transmitted The final answer to control of VES is a con tinuing expansion of our knowledge re garding all phases of the disease, and in telligent and vigorous prosecution of the measures already taken

currence of a virus capable of assuming a different antigenic make up whenever the biological environment becomes favorable or necessary for a new type to appear

CONTROL

The ability to control VES depends, as it does for all diseases, on a basic knowl edge of the modes of transmission Cur rently such modes include direct contact and the feeding of raw garbage It is neces sary, therefore, to interrupt the known infection chains, and this, in essence, is the history of VES control efforts

In California prior to 1954, eradication and quarantine were used to break the direct contact links In 1932 and 1933, the time honored methods of slaughter and dis infection so successfully employed against FMD in this country were applied, but the disease reappeared in 1934, four hundred to five hundred miles distant from the first two foci In 1934, slaughter measures were abandoned, and a quarantine of infected ranches was imposed instead This quar antine consisted of embargoes against moving swine from infected premises until all signs of the disease had disappeared In addition, the movements of vehicles and men were controlled to minimize the pos sibility of spread by this route After quar antine was imposed, a differential diag nosis between FMD, VS and VES was made In stockyards under quarantine, af fected hogs were released for slaughter in accordance with the meat inspection regu lations governing each vesicular disease Duckworth (1953a), questioning the value of restrictive quarantine, slaughter, and disinfection in California, concluded that these methods of eradication were not likely to succeed unless the disease was attacked at its source

The appearance of the virus outside the confines of California, in 1952, set in mo tion the control measures immediately available In brief, these consisted of the following (1) federal quarantines re stricting the interstate movement of swine and pork products from infected areas, (2)

cleaning and disinfection of railroad cars, feed, water, and rest stations contaminated by infected swine, and (3) closing and disinfection of all suspected stockyards, and close inspection of all animals coming in for slaughter (Agricultural Research Service Report, 1952) In August, 1952, the Secretary of Agriculture declared a national emergency This act made federal funds available to carry out an active eradication program, including the slaugh ter of infected hogs and the payment of indemnities in the states that were able to match federal funds for such payments

During the first year of the program, some 180,000 swine were killed and the quarantine measures vigorously enforced In spite of this, the disease spread so rapidly that, within this same period, it appeared in 40 states and the District of Columbia, being reported from 50 grain fed herds, 522 garbage fed herds, and 234 serum plants, stockyards, and picking plants Ultimately the infection was re ported from a total of 42 states and the District of Columbia

Throughout the history of the disease in California, the role of raw garbage as a means of perpetuating the disease had been recognized Federal, state, and local live stock sanitation officials had urged the pas sage of legislation to prohibit feeding raw garbage to swine (Wright, 1913, Duck worth and Traum, 1951, Shope et al, 1952, Mulhern, 1953, Duckworth, 1953b)

The national outbreak of VES in 1952 served to focus attention upon the need for adequate legislation to control this antiquated practice Prior to 1952, vir tually no garbage fed to swine was cooked In June, 1953, 35 states had adopted garbage cooking regulations, by September, 1953, 46 states required the cooking of garbage, and as of January 1, 1958, only Connecticut had not passed legislation to that effect

The passage of such legislation in the various states permitted certain additions and modifications in the original control program Perhaps the most important was

——— 1933b Report Chief Bur Anim Ind , U S Govt Print Office, Washington, D C
MOTT, L O   1956   Epizootiology of Vesicular Exanthema of swine Proc Symposium on
    Vesicular Diseases Agr Res Serv U S D A 74
——— AND PATTERSON, W C   1956 Personal communication
———, ———, SONGER, J R. AND HOPKINS S R   1953   Experimental infections with Vesicular
    Exanthema Proc. 57th Ann Meet U S Livestock San Assn 334 1 II
MULHERN, F J   1953   Present status of Vesicular Exanthema eradication program   Proc 57th
    Ann Meet U S Livestock San Assn 1
———   1956 Personal communication
——— AND PATTERSON, W C   1956 Yearbook of Agr U S D A p 369
MYERS, L P   1955   Vesicular Exanthema control program Bull Calif Dept Agr 45 73
OLITSKY, P K , TRAUM, J AND SHOENING, H W   1928 Report of the Foot and Mouth disease
    commission of the U S D A Tech Bull 76 1
PATTERSON, W C , AND SONGER, J R   1954 Experimental infections with Vesicular Exanthema
    of swine Part III viremia studies in swine and their relationship to vesiculation Proc
    58th Ann Meet U S Livestock San Assn 396
REPPIN, K. AND PYL, G   1935   Maul und Klauenseuche oder stomatitis vesicularis Arch wiss
    prakt Tierheilk 68 183
SELLERS R F   1955   Growth and titration of the viruses of Foot and Mouth disease and Vesicu
    lar Stomatitis in kidney monolayer tissue cultures Nature 176 547 Sept 17
SHOPE, R E   1955   Epizootiology of virus diseases Advances in Vet Science Academic Press Inc
    New York 2 1
———, SUSSMAN O AND HENDERSHOT R A   1952   Administrative considerations of garbage
    feeding with reference to Vesicular Exanthema and Trichinosis Proc 56th Ann Meet
    U S Livestock San Assn 218
SIMMS B T   1953 Progress made in eradication of Vesicular Exanthema Report Chief, Bur
    Anim Ind 1 2 118
SPECIAL REPORT JOINT LEGISLATIVE COMMITTEE ON AGRICULTURAL AND LIVESTOCK PROBLEMS 1955
    Published by the Senate State of California, p 102
TRAUM J   1933 Foot and Mouth disease differential diagnosis Proc 5th Pacific Sci Congress
    Canada 4 2907
———   1934 Foot and Mouth disease Specific treatment eradication and differential diag
    nosis Proc. 12th Internat Vet Cong 2 87
———   1936 Vesicular Exanthema of swine Jour Amer Vet Med Assn 88 316
———, AND WHITE, B W   1911 Unpublished data
WICKTOR, C E , AND COALE, B B 1958 Vesicular Exanthema Vet Med 53 516
WRIGHT, W H   1943 Health problems concerned in the disposal of garbage by feeding it to
    swine Amer Jour Public Health 33 208

REFERENCES

Agricultural Research Service Report 1952 Report of developments on eradication of Vesic ular Exanthema 1 1 USDA

—— 1954 Losses in Agriculture A preliminary appraisal for review 131 USDA

Bankowski, R A 1954 Personal communication

——, Keith, B, Stuart, E E, and Kummer, M 1954 Recovery of the fourth immunological type Vesicular Exanthema virus in California Jour Amer Vet Med Assn 125 383

——, and Kummer, M B 1955 Vesicular Stomatitis and Vesicular Exanthema differentiation by complement fixation Amer Jour Vet Res 16 374

——, Perkins, A G, Stuart, E E, and Kummer, M 1955 Epizootiology of Vesicular Exanthema in California Proc 59th Ann Meet U S Livestock San Assn, p 356

—— —— and 1956 Recovery of new immunological types of Vesicular Exanthema virus Proc. 60th Ann Meet U S Livestock San Assn, p 302

——, and Pfeiffer R W 1955 Cultivation of Vesicular Exanthema virus in vitro using pig embryo tissues Proc Soc Exper Biol and Med 88 2, 209

——, Wichmann, R, and Kummer, M 1953 Complement fixation test for identification and differentiation of immunological types of the virus of Vesicular Exanthema of swine Amer Jour Vet Res 14 145

——, and Wood, Margaret 1953 Experimental Vesicular Exanthema in the dog Jour Amer Vet Med Assn 123 115

British Report 1937 Fifth Prog Report Foot and Mouth Disease Research Committee, Brit Min Agr and Fisheries 99

Brooksby, J B 1954 Étude expérimentale de l Exanthema Vésiculeux Rep Off int Epiz, 22 1

Bureau of Animal Industry Order No 383 (Rev) 1953 Part 76, Regulations restricting inter state movement of swine and certain swine products because of Vesicular Exanthema USDA

Chow, T I Hanson, R P, and McNutt, S H 1951 The pathology of Vesicular Stomatitis in cattle Proc. Book Amer Vet Med Assn, p 119

Crandill, R and Madin, S H 1957 Unpublished data

Crawford A B 1937 Experimental Vesicular Exanthema of swine Jour Amer Vet Med Assn 90 380

Duckworth C U, and Traum J 1951 Foot and Mouth disease and other diseases of animals of Europe Special report State Senate Interim Committee on Livestock Diseases Published by the Senate State of California 1

——, and White B B 1943 Twelve years of Vesicular Exanthema Proc 47th Ann Meet U S Livestock San Assn, 79

Duckworth C E 1953a Vesicular Exanthema of swine Bull Calif Dept Agr 42 1

—— 1953b. Special Report Joint Legislative Committee on Agricultural and Livestock Problems Published by the Senate, State of California

Dulbecco, R 1952 Production of plaques in monolayer tissue cultures by single particles of an animal virus Proc Nat Acad Sci 38 747

Galloway, I A, and Nicolau, S 1928 Histological study of the development of Foot and Mouth disease in the tongue of the guinea pig rabbit and ferret Third Prog Report, Foot and Mouth Disease Research Committee, Brit Min Agr and Fisheries, App III, 104

Hurt, L M 1940–41 Report Los Angeles County Livestock Department 28

Long, J D and Johnson, C C 1952 Equipment for the heat treatment of garbage to be used for hog feed Issued jointly by USDA and U S Dept Health, Educ, and Welfare

—— and 1954 Equipment for the heat treatment of garbage to be used for hog feed Issued jointly by USDA and U S Dept Health, Educ, and Welfare Supplement No 1, February

McClain, M E, Hackett, A J, and Madin, S H 1958 Plaque morphology and pathogenicity of Vesicular Exanthema virus Science. 127 1391

——, Madin, S H and Andriese P C. 1954 In vitro cultivation and cytopathogenicity of Vesicular Exanthema virus Proc. Soc. Exper Biol and Med 86 771

Madin, S H 1954 Preliminary studies on the prophylactic value of type A Vesicular Exanthema immune serum Jour Amer Vet Med Assn 125 47

—— 1956a Unpublished data

—— 1956b Preliminary studies on the prophylactic and therapeutic value of type B Vesicular Exanthema immune serum Jour Amer Vet Med Assn 129 568

——, Andriese, P C, and Darby, N 1958a Behavior of Vesicular Exanthema of swine virus in tissue culture Unpublished data

——, Kniazeff, A F, and Darby, N 1958b Immunology of Vesicular Exanthema of swine 1 Diagnosis and differentiation from Vesicular Stomatitis by serum neutralization and in vitro host susceptibility In preparation

——, and Traum, J 1953 Experimental studies with Vesicular Exanthema of swine Vet Med 48 395, 413

——, and —— 1955 Vesicular Exanthema of swine Bact Rev 19 6

Mohler, J R 1953a Personal communication

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Vesicular Stomatitis

Vesicular stomatitis, first known as a dis ease of horses and later as one of cattle and swine, also affects man and several wild animals In the United States, farmers call it "sore nose" or "sore mouth" and in South America and Mexico they know it as pseudoaphtosa and mal de yerba Vesicular lesions are produced by the virus in the mucosal tissue of the mouth and in the skin of the coronary band of the foot of the horse, cow, pig, and deer Man, raccoon, and bobcat have an inapparent or febrile disease A number of animals can be experimentally infected A fatal encephalitis is induced in the mouse, guinea pig, hamster, and ferret Several investigators have found the goat, sheep, rabbit and dog to be refractory to inoculation The disease appears to be limited to the western hemisphere, where it is enzootic on the coastal plain of the region extending from the Carolinas southward around the Gulf of Mexico and the Caribbean Sea Less frequently, epizootics occur south to Peru and Argentina in South America and north into Canada in North America

ETIOLOGY

Vesicular stomatitis of swine, cattle, and horses is caused by two antigenically distinct viruses, the New Jersey type and the Indiana type, which are similar in size and pathogenic potential and probably phylogenetically related (Cotton, 1926, 1927)

Galloway and Elford (1933) first estimated the size of Indiana and New Jersey type vesicular stomatitis virus to be 70 to 100μ by passing vesicle fluid from guinea pig foot pads through graded collodion membranes Bauer and Cox (1935) confirmed the report of Galloway and Elford with mouse adapted and tissue culture propagated strains of vesicular stomatitis virus Later, Elford and Galloway (1937), using the sedimentation method, calculated the size of vesicular stomatitis virus, Indiana type, to be 74μ Chow and associates (1954) examined embryo propagated virus in the electron microscope (Fig 11 1) The particles were rod shaped and averaged 60μ in diameter and 210μ in length Bradish and his associates (1956) have described a small complement fixing, non infective particle which was associated with the larger, infective particle of vesicular stomatitis virus

Studies of the physical properties of vesicular stomatitis virus and its cultural characteristics have been conducted in 4 laboratory hosts — guinea pig, mouse, chicken embryo, and several types of tissue culture A small amount of virus inoculated into the foot pad of the guinea pig usually results in development of a vesicle within 18 hours (Cotton, 1926) The pathogenesis is similar whether the virus is introduced into the pad epithelium of the guinea pig or the snout epithelium of the pig Usually

tured virus equals the initial titer of the culture (Timm, 1955).

Vesicular stomatitis virus is resistant to pH changes and has a moderate resistance to heat and to chemical inactivation. With the chicken embryo and the mouse as indicator hosts and using both the New Jersey and Indiana types of vesicular stomatitis virus, Fong and Madin (1954) have shown that the vesicular stomatitis virus has an unusually wide range of stability to pH. The virus tolerated a pH as low as 4 and as high as 10 for one hour with only a moderate diminution of titer. The virus remains active for 1 to 3 weeks at room temperature, depending on suspending medium. Frozen cultures may be stored for several years without great loss of activity. English workers have found the virus to be sensitive to the action of visible light, but this was not confirmed by Madin (1952). Ultraviolet light inactivates the virus rapidly. The virus is most stable in serum or thioglycollate broth. Although it remains active for a time in sterile water, it appears to be quickly inactivated by dilution in physiological saline (Brandly et al., 1953).

CLINICAL SIGNS

Swine may be infected by ingesting the virus or by the virus entering through abrasions in a susceptible area of the epithelium. The first sign of disease is an increase in body temperature which occurs 24 to 48 hours following infection. The temperature will range between 104° and 107° F, rarely going to 108°. Drooling may be the next sign. The vesicles appear on the tongue, snout, or coronary band 18 to 72 hours after infection They originate as papules which are rarely seen and rapidly form into blebs filled with clear liquid and varying in diameter from a few millimeters to 3 centimeters On the snout, vesicles are more delicate and often larger than on the coronary band Vesicles may persist as long as 21 hours, although usually they rupture within a few hours after development. The temperature then falls rapidly to normal. A few animals may go off feed but most of them retain their

TABLE 11.1
Stability of Vesicular Stomatitis Virus
(Virus active for time indicated)

Reference[*]	Preparation	Temperature, C.					
		60°	55°	37°	25°	4°	−20°
1	VSV pad†	0	30 min.	4 da		40 da	
3	VSV pad	.				31 da	
1	VSV pad (crystal-violet‡)					8 da	
1	VSV pad (phenol 0 5%)			1 da.		23 da	
2	VSV pad (sodium bifluoride 0 01%)	. .			24 hr		
2	VSV pad (sodium silicofluoride 0 01%)	. .			24 hr		
3	VSV pad (ethyl alcohol 60%)	.			24 hr		
3	VSV pad (cresol 3%)	.			6 hr		
3	VSV pad (sodium hydroxide 2%)				1 min		
3	VSV pad (glycerol 50%)	.				135 da	
4	VSV egg§	30 min.	30 min.				
5	VSV egg	0	15 min.	5 da			235 da

* References
  1 Shahan, 1946 Amer Jour. Vet. Res. 7 27.
  2. Manthei and Eichhorn, 1941. Jour. Agr. Res. 63 41.
  3 Olitsky, 1927 Jour Exper Med. 45 969
  4 Madin, 1952. ONR Report, University of California.
  5 Brandly et al., 1953. Camp Detrick Report, University of Wisconsin
† VSV pad = NJ type VSV guinea pig foot pad origin.
‡ Crystal-violet as contained in hog cholera vaccine.
§ VSV egg = NJ type VSV allantoic fluids of embryonating egg

early on the second day after inoculation, small red points of swelling appear and rapidly develop into fluid filled blebs or vesicles The virus reaches a titer of about 10^4 in the fluid and in the epithelium covering it before the vesicle ruptures, or ten a few hours after its formation Healing is accomplished within 7 to 10 days

Mature mice inoculated intracerebrally develop a fatal encephalitis The first sign is usually hypersensitiveness, followed by tremors, ataxia or spastic paralysis of the posterior extremities, and death in 3 to 5 days (Cox and Olitsky, 1933) The suckling mouse is more sensitive than the adult mouse, becoming fatally infected by intranasal and intraperitoneal inoculation as well as intracerebral injection Between the 21st and 35th day, mice change from a state of nearly 100 per cent susceptibility to extraneural infection to complete refractoriness (Sabin and Olitsky, 1937) In tracerebral susceptibility remains through out the life of the mouse The virus grows rapidly in the brain and titers of 10^5 to 10^7 virus units per gram of brain material are frequently obtained (Madin 1952)

The chicken embryo may be inoculated on the chorio allantoic membrane or in the allantoic chamber but the virus is usually cultivated by the latter route (Burnet and Galloway, 1934 Brandly et al, 1953) The virus grows rapidly and the embryo is killed within 24 to 48 hours At death the embryo is congested and the allantoic fluids and amniotic fluids are clear The membrane usually shows little change Sigurdsson (1943) found that growth of virus was favored in embryos of 7 days as compared to 10 days and at low incubation temperatures of 35 to 36° C as compared to 39 to 40° C This was indicated by greater lethality for the embryo and in creased production of virus Madin (1952) confirmed the observation when he titrated virus in embryos of several ages and found that susceptibility was inversely related to age 7 day, 10^8, 9 day, $10^{5.5}$, 11-day, $10^{4.5}$ and 13-day, 10^{0}

Vesicular stomatitis virus has been grown in tissue cultures prepared from the epithelial cells of bovine tongue, pig embryo pig kidney, guinea pig kidney and chicken embryo Suspended cell and monolayer preparations have been used With the first culture, growth of the virus is detected by pH change and by subinoculation in a susceptible host With the monolayer culture, cytopathogenic changes and development of plaques are observed between 24 and 72 hours after inoculation (Bachrach et al, 1955 Fellowes et al, 1956) The virus is adsorbed very rapidly by the cells of the tissue culture There is an immediate eclipse phase partly obscured by a slight increase in the virus an hour after inoculation It takes between 7 and 8 hours before yield of tissue cul

FIG 11 1—Electron micrograph of vesicular stomatitis virus (New Jersey type) Virus propagated in chicken embryos purified by ultracentrifugation and shadowed with uranium

polymorphonuclear leukocytes Most of the leukocytes are caught as part of the infiltrates within the tissue of the base of the vesicles The epithelial cells, after disarrangement by intercellular edema, suffer degenerative changes characterized by disappearance of the protoplasmic intercellular bridges and the gradual diminution of cytoplasm The nuclear shrinking, pyknosis and karyolysis then become evident The process extends downward into the dermis, the basal cell layer is disarranged and infiltrated Inflammation, including congestion of the dermis, sometimes penetrates deep in the dermal region Edema and hemorrhage in the dermal papule, engorgement of the lymph vessels and blood vessels, and perivascular leukocytic infiltration are usually observed The normal appearance of sebaceous glands is retained Sweat glands are not affected although sometimes there is hemorrhage around the hair shaft With the exception of the liver, where congestion is sometimes apparent, significant lesions are not seen in other tissues such as brain, muscle, lungs, heart, spleen, intestines, kidneys, adrenals, pancreas, and lymph glands

DIAGNOSIS

The primary problem in diagnosis of vesicular stomatitis is to differentiate it from the diseases produced by the viruses of foot and mouth disease and vesicular exanthema Certain infectious and noninfectious conditions such as mucosal disease, blue tongue, and caustic poisonings can be confused with it. Vesicular stomatitis is readily identified by isolation of the virus in guinea pig foot pad, chicken embryo, or mouse brain, or by transmission to a susceptible horse Identification by isolation or transmission is dependent primarily upon obtaining a satisfactory sample from the suspect animal and upon the inoculation of known susceptible hosts Difficulties involved in getting susceptible horses in an enzootic area have delayed and jeopardized the diagnosis of the disease Isolation in guinea pigs, chicken embryos, and mice sometimes poses a problem of adaptation of the virus to the experimental

host Henderson (1948) reported difficulty in infecting chicken embryos with virus from a bovine epithelium preparation which was readily infective for guinea pigs This may be rare, as 6 lots of vesicular materials obtained from swine and cattle during the 1955 season in southeastern United States were readily established in embryonating eggs and adult mice (Karstad et al, 1956) Fellowes and co workers (1956), using bovine epithelium virus of New Jersey and Indiana type, titrated the virus in 8 day chicken embryos, 3 to 4 week old mice, adult guinea pigs, guinea pig kidney tissue culture, calf and adult bovine tongues Chicken embryos and mice were the most sensitive, New Jersey type virus being detected more readily in the embryos, and the Indiana type more readily in mice Tissue culture and bovine tongues were comparable to each other and of intermediate sensitivity Guinea pig foot pad and calf tongue were the least sensitive Specific pathology does not differentiate vesicular stomatitis from foot and mouth disease or vesicular exanthema (Chow and McNutt, 1953) It may differentiate vesicular stomatitis from certain other noninfectious conditions if representative material is made available for examination

Considerable work has been done on the serology of vesicular stomatitis Recovered animals possess antibodies detectable by serum neutralization test (Long and Olitsky, 1928, Brandly et al, 1951) and by complement fixation tests (Brooksby, 1948, Binkowski and Kummer, 1955) Both the neutralization test and the complement fixation test are useful in epizootiological surveys The complement fixation test is the less expensive and very rapid, but can be used only with sera whose anticomplementary activity is known Advantages and the limitations of both tests suggest that serum neutralization tests will be used in surveys of the reservoir potentials of wild life or for the detection of long persisting antibody (Karstad et al, 1956), and the complement fixation test where a rapid, inexpensive procedure is needed to determine the disease status of cattle and swine

appetites even when eating becomes pain ful because of lesions on the tongue or when there is difficulty of movement be cause of lesions of the feet Although the formation of vesicles on the skin of the lips snout coronary band and interdigital spaces is the most characteristic sign of the disease not infrequently the erosion rather than the vesicle is seen, as exfoliated tissue remains adherent to the margins of the lesion and this stage persists for as long as a week Involvement of the coronary area sometimes results in loosening or sloughing of the claws Sick animals may be reluctant to stand and may move with stiffness which suggests a degree of pain Animals resist being touched, as early as the papular stage Duration of sickness is about 2 weeks if there are no complications Prognosis is good Scars do not remain after healing

Inapparent cases of disease occur among swine, particularly in animals that ingest the virus without the virus coming in con tact with abrasions in the mucosal mem branes Such animals may show a thermal response which however, would rarely be detected Whether a few or many of the swine show clinical signs of vesicular stoma titis depends upon the epizootiological situation

PATHOLOGICAL CHANGES

The macroscopic lesion of the disease is the vesicle and it appears in the epithelial tissue (Chow and McNutt, 1953) The first sign of change is spongiosis shown by the loosening of the epithelial cells of the Mal pighian layer (Fig 11 2) The intercellular bridges between the prickle cells are stretched by the edematous fluid which ac cumulates in the intercellular spaces This results in the formation of small vacuoles among cells A large number of small vacuoles produce the swelling apparent on the surface as the papule Confluence of neighboring vacuoles produces a vesicle which appears from the epithelial surface as an almost transparent bleb Microscopi cally, there is a multilocular intercellular edema scattered throughout the Malpighian layer which has increased in thickness Spongiotic epithelial tissue and some keratinized cells lie atop the vesicle The fluid of the vesicle contains relatively few

FIG 11 2—Spongiosis of the snout epithelium at early stages of vesicular stomatitis infect on X 350

In the western hemisphere, there are regions in which vesicular stomatitis is enzootic and other regions into which it extends occasionally as an epizootic (Hanson, 1952) The disease has been reported from Panama, parts of Central America, Mexico (Camargo, 1954), about half of the United States and one of the Canadian provinces in North America (Hanson, 1952), and in South America it was described in Colombia (Reyes, 1946), Ecuador (Rosero Sanchez, 1952), Venezuela (Gallo et al, 1950), Peru (Strozzi and Ramos Saco, 1953), and Argentina The disease reappears each year in the enzootic regions, and in the epizootic regions the disease occurs less frequently, perhaps once in 10 years

In the enzootic areas, high levels of antibodies are found in most susceptible animals (Adams et al, 1956) In the enzootic area of Georgia 100 per cent of the horses and about 50 per cent of the cattle and swine carried antibodies In the epizootic area, antibodies are found only in animals of the age group that went through the previous epizootic

Enzootic vesicular stomatitis exists in Colombia and Venezuela on the Caribbean, and in Mexico on the Gulf of Mexico In the United States it is found in Georgia, South Carolina, and Florida on the Atlantic Coastal Plain, and probably also in Alabama, Louisiana, and Texas on the Gulf of Mexico There is some evidence that an enzootic focus may exist in the Colorado and New Mexico areas of the southern Rockies

Epizootic vesicular stomatitis has appeared in the Rocky Mountain states — Colorado, Utah, Montana, in the upper Mississippi Valley — Wisconsin, Minnesota, Manitoba, and in the Appalachian area — Kentucky, Tennessee, North Carolina Virginia, and West Virginia The outbreak in Argentina was probably also an epizootic

Infection of swine has been reported principally in the enzootic areas of Georgia, North Carolina, South Carolina, and Louisiana, and in Colombia and Venezuela (Schoening, 1954) The most important exception was the first outbreak of vesicular stomatitis in swine to be reported in the United States, which occurred in August, 1943, in Missouri (Schoening, 1943) About half of 1,500 swine in a hog cholera serum plant were involved The disease was severe and characterized by pyrexia, lameness, and a few deaths Virus of the New Jersey type was isolated from the swine Investigation did not reveal how the virus got into the plant, but within the plant, virus was apparently spread both by inoculation procedures used in hyperimmunization of swine and by contact. The older and heavier swine showed the more severe reactions

Heiny (1945) reported that in 1944 the disease was seen in swine in Colorado The virus was not isolated from hogs in this outbreak but it was isolated from cattle in the same area and was shown to be New Jersey type virus An outbreak of vesicular stomatitis in swine was reported from Georgia in May, 1952 It continued through the summer into August Cases have been reported each succeeding year The first positive diagnoses were made in 1952 on 3 premises, involving over a thousand head of swine, and in 1953 on 10 premises, involving 700 head of swine Vesicular stomatitis was then reported in Wayne County, North Carolina in May, 1953, in Beaufort County, North Carolina in August, 1954, in Rockingham County and Shenandoah County, Virginia, in September, 1953, in Holmes County, Florida, July, 1954, in Saint Laundry, Louisiana, in August, 1954 (Schoening 1954)

The geographical distribution of the disease and particularly the limitation of the enzootic disease to southern areas suggest that environmental factors have a considerable importance Epizootic vesicular stomatitis occurs principally in August and September, though the first cases may be seen in June and the last in October Cases in the enzootic areas as in Georgia, are seen from the last of May to the latter part of October (Adams et al, 1956) The incidence of disease corre-

TREATMENT

There is no specific treatment for vesicular stomatitis of swine. Abundance of water and soft feed should be kept before the animals to avoid an excessive loss of weight during the period of fever and to reduce injury to the mucosal tissues. Secondary infections which may occur in the abraded tissues should be treated according to the type of organism involved.

IMMUNITY

Within 10 days to 2 weeks after the development of an infection in swine, neutralizing and complement fixing antibodies may be detected in the blood stream. The titer of both increase until the fourth or fifth week and then persist at high level for several months before gradually falling (Sorensen, 1953). The complement-fixing titer disappears long before the neutralizing titer (Mott, 1956). Swine are usually refractory to re-exposure a month after infection. However, this immunity may be broken by introducing a large quantity of virus on the mucosal tissues. It appears probable, on the basis of information from the enzootic area in Georgia, that natural reinfections of swine do not occur. The persistence of neutralizing antibodies in swine and the duration of refractoriness requires further study since the detection of neutralizing antibodies in isolated cattle 7 years after infection. Suckling pigs receive antibodies from the sow. In one area in Georgia, seventy-one per cent of the pigs which were 3 months of age or younger had antibodies. This suggests one reason for refractoriness of young pigs in some areas.

Pigs that are immune to the Indiana type of vesicular stomatitis are readily infected by virus of the New Jersey type, and the reverse is true. There is no cross-immunity between the two virus infections.

EPIZOOTIOLOGY

While more is known about the epizootiology of vesicular stomatitis than about most diseases of livestock, a considerable portion of the story is yet to be learned.

The disease has a restricted geographical distribution, a seasonal appearance, and a host range that is known to include both domestic and wild animals. The means of transmission has not been established.

Vesicular stomatitis is a disease of the western hemisphere (Fig. 11.3). Stomatitis of horses was described in the United States as early as 1821 and since then has been reported at intervals (Hanson, 1952). It is probable that the disease has always been present on this continent. The 1915—16 outbreak in Europe was traced to animals imported from the United States and Canada (Jacoulet, 1915). The 1898 epizootic in South Africa described by Theiler (1901) may also have been an introduction from the western hemisphere, as veterinarians in Africa and in Europe have not seen the disease since that time. Stomatitis of horses has been observed occasionally in countries of Asia, but there is no evidence that it is caused by the virus of vesicular stomatitis.

EPIZOOTIC AREAS
1 WIS, MINN, KAN 1926, '37, 40
2 N.M., COL., UTAH, MONT. 1926, '35, '37, '42, 44
3 TEXAS 1926, '41, '44, '49
4 NO, WVA, VA. 1899, 1916, '24, '36

ENZOOTIC AREAS
5 COASTAL PLAIN GA., S.C., FLA.
6 MEXICO
7 VENEZUELA, COLOMBIA
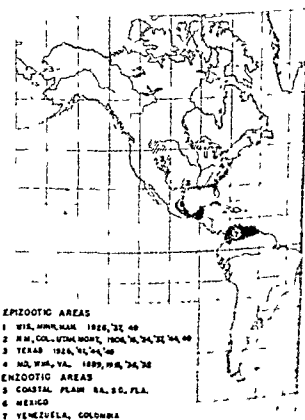
FIG. 11.3—Geographical distribution of vesicular stomatitis.

ever, were not made more susceptible by a deficient diet

Vesicular stomatitis of horses and cattle was recognized years before the disease was reported in swine. Did this mean that a mutation occurred and a new strain with a predilection for swine was introduced or that there was inadequacy of reporting? Farmers and veterinarians in the Wisconsin Minnesota Manitoba outbreak in 1949 did not observe any instance of vesicular disease in swine. The apparent absence may not be of great significance, however as the region has a small pig population, and swine on the premises where the disease existed in cattle and horses were not tested for antibodies. The picture is similar in the northern Rocky Mountain region. Disease of swine could have been overlooked in these areas. Vesicular stomatitis was not uncovered in Georgia until the vesicular exanthema eradication campaign of 1952. Veterinarians and farmers in the area assured investigators that the disease was not new, that it certainly had been in the area for over 50 years (Adams et al, 1956).

Supposing that vesicular stomatitis in swine has existed whenever the disease has occurred in an area in which there was a large swine population, the possibility that there are strains with an affinity for swine is still unsettled. Six strains of virus isolated from swine and cattle in the 1952-54 period differed slightly among themselves in pathogenicity for swine and laboratory animals but this was not correlated with the species of origin. There is no evidence that swine strains of vesicular stomatitis have developed as they have for foot and mouth disease.

Each summer vesicular stomatitis has reappeared in the United States after being quiescent from November to May. Where does the virus hide during the winter season? Certainly not outside of living cells on the walls of pens or piles of hay as it is too unstable, apparently not in domestic animals that have had the disease, as they develop good antibody, perhaps, then, in some wild species. A search for the reser-

voir in southeastern Georgia where the disease is enzootic revealed that wild animals do become infected. About 60 per cent of the deer and 45 per cent of the raccoons and bobcats possessed neutralizing antibody indicating prior infection (Karstad et al, 1956). Experiments in which these animals were exposed to the virus resulted in a mild acute infection, a short carrier period, and a rapid and strong immunity. There was no suggestion of a carrier state (Adams et al, 1956). Man is also susceptible and develops an influenza like infection following exposure. The initial cases to be described were laboratory accidents (Hanson et al, 1950. Fellowes et al, 1955), but in 1956 a survey of farm families in southeastern Georgia revealed that the virus infected man in rural communities. Although the host range of vesicular stomatitis has been extended in the last few years and may be extended further, chronic or carrier type infection has not been demonstrated in any warm blooded animal. Latency cannot be excluded, however, serum neutralization titers persist without diminution in isolated cattle for 7 years. The possibility of virus persisting in a parasite or some in vertebrate has been studied only superficially but appears more promising as a solution to the survival of vesicular stomatitis over the winter (Adams et al, 1956).

Vesicular stomatitis appears to be enzootic in the warmer portion of the western hemisphere. When conditions are favorable, it spreads throughout suitable areas in the cooler regions of northern United States and southern South America. As long as there is a reservoir of the virus and a suitable vector, the virus will continue to persist irrespective of quarantine. It is possible that better knowledge of the epizootiology would make control possible through management of the reservoir or vector. If it were desirable, cattle and horses probably could be rendered immune by intramuscular inoculation with strains of virus now available. It is very doubtful that such strains could be used in swine without generalization of the disease. Im-

sponds fairly well with the activity of mosquitoes and biting flies It has not been possible to show relationship between wet years and epizootic years For example, 1926, 1937, and 1949 in Wisconsin were not similar in the rainfall Other years occurring in the interim were wetter or dryer The vector picture, however, is much more complicated than total rainfall The pattern of rainfall, heavy showers that leave breeding pools or heavy rains at critical periods, are more significant than the total rainfall

Roberts and associates (1956) showed that populations of potential vectors in the Wisconsin epizootic area vary from year to year Some years, a particular species of mosquito would occur in large numbers in the spring In other years the same species would be found in the fall Some species increased in numbers over a period of years and others decreased Vectors furthermore, were not evenly distributed throughout an area as small as a county Wet land pastures had different species and different populations than dry land pastures Tree cover or lack of tree cover modified the picture Insects found in the stable differed from those found in the pastures. It is significant that numerous observers in widely separated regions have *reported the prevalence of the disease in* pastured animals and its rarity in stabled animals, its prevalence in animals in wooded pastures and its rarity in open pastures, its prevalence in animals on wet land pastures and its rarity on dry land pastures

The susceptibility of the animal cannot be ignored Age certainly is a factor Mature animals appear to develop vesicular stomatitis more readily than immature animals Veterinarians observed thousands of cattle with lesions of vesicular stomatitis during the 1949 Wisconsin epizootic (Brandly et al, 1951), only one animal less than six months of age was reported to be diseased, although immature animals must have made up a considerable part of the herd at risk Experimentally, calves may be infected, the disease, however, develops

more slowly and lesions are milder A biphasic rather than a monophasic temperature curve has been found Fellowes and co workers (1956) obtained higher titers in adult bovine tongue epithelium than in the tongue epithelium of calves Most observers have found the larger and older pigs more susceptible than the smaller and younger ones Sanders and Quin (1944) reported that in the Missouri outbreak the disease was limited, with one exception to the heavy, hyperimmune, garbage fed hogs In studies of experimental transmission Shahan (1946) found the older pig to be more susceptible than the young pig On the other hand, Wagener (1932) reported that vesicular stomatitis can readily be transmitted by direct contact among young swine and less readily among older swine Schoening (1954) cited an outbreak in Wayne County, North Carolina, in which a brood sow and 5 of her suckling pigs showed lesions Lesions were not found in the 10 feeder shoats or 1 boar which were also in association with the sow and her pigs

Some of the basic work on susceptibility and age was conducted with vesicular stomatitis in laboratory animals Sabin and Olitsky, (1937) found mice of all ages to be susceptible if the virus was *introduced directly into the brain* Young mice, from birth to about one week after weaning also could be infected if the virus was placed on the nasal epithelium or injected intramuscularly or intraperitoneally In laboratory animals as in livestock the best epithelial lesions have been obtained in mature animals, large guinea pigs being more satisfactory than small guinea pigs for foot pad inoculation Environmental factors affect the host response Mice subjected to certain changes in environmental temperature died in greater numbers— 60 per cent of those stressed as compared to 12 per cent of those not so stressed (Griffin et al, 1951) Nutrition may contribute to the total response Sabin (1941) found that the resistance to intranasal infection could be delayed in mice on a deficient diet Mice once resistant how

———— RASMUSSEN A F BRANDLY C A AND BROWN J W   1950 Human infection with the virus of vesicular stomatitis Jour Lab and Clin Med 36 754

HEINY, E   1915 Vesicular stomatitis in cattle and horses in Colorado No Amer Vet 26 726

HENDERSON W M   1948 Some observations on the quantitative study of vesicular stomatitis virus Jour Comp Path 58 172

JACOULET M   1915 Au sujet d une stomatite erosive de nature indéterminée (chez le cheval) Bull Soc cent Med Vet 68 576

KARSTAD L H ADAMS E V HANSON R P AND FERRIS D H   1956 Evidence for the role of wildlife in epizootics of vesicular stomatitis Jour Amer Vet Med Assn 129 95

LONG P H AND OLITSKY P K   1928 Immun ty in guinea pigs to the virus of vesicular stomatitis Proc Soc Exper Biol and Med 25 478

MADIN, S H   1952 Vesicular diseases Report to the Office of Naval Research Univ of Calif Mimeo

MANTHEI C A AND EICHHORN A   1941 Use of sodium bifluoride and sodium silicofluoride in the disinfection of hides Jour Agr Res 63 41

MOTT L   1956 Personal communication

OLITSKY P K   1927 Physical chemical and biological studies on the virus of vesicular stomatitis of horses Jour Exper Med 45 969 Also stud es from Rockefeller Inst Med Res 63 1

———— AND LONG P H   1928 Histopathology of experimental vesicular stomatitis of the guinea pig Proc Soc Exper Biol and Med 25 287

———— TRAUM J AND SCHOENING H W   1926 Comparative stud es on vesicular stomatitis and foot and mouth diseases Jour Amer Vet Med Assn 70 147

REYES H A   1946 Diagnóstico de la cepa EVF como virus de la estomatitis vesiculosa Rev Med Vet (Bogota) 15 57

ROBERTS R H DICKE R J HANSON R P AND FERRIS D H   1956 Potential insect vectors of vesicular stomatitis in Wisconsin Jour Infect Dis 98 121

ROSERO SANCHEZ C   1952 Campa a contra la estomatitis vesiculosa Consorcio de Cent Agr de Mambi Rev Ecuador 70 13

SABIN A B   1941 Constitutional barriers to involvement of the nervous system by certain viruses with special reference to the role of nutrition Jour Pediatrics 19 596

———— AND OLITSKY P K   1937 Influence of host factors on neuroinvasiveness of vesicular stomatitis I Effect of age on the invasion of the brain by virus instilled in the nose Jour Exper Med 56 15

SANDERS L F AND QUIN A H   1944 Vesicular stomatitis in swine Report of a naturally occurring outbre k Its differentiation from foot and mouth disease and vesicular exanthema No Amer Vet 25 413

SCHOENING H W   1913 Vesicular stomatitis in swine Proc 47th Ann Meet U S Livestock San Assn p 85

———— 1954 Vesicular stomatitis in swine Proc U S Livestock San Assn 58 390

SELLERS R F   1955 Growth and titration of the viruses of foot and mouth disease and vesicular stomatitis Nature (London) 176 547

SHAHAN M S   1946 Effect of temperature phenol and crystal violet on vesicular stomatitis virus Amer Jour Vet Res 7 27

———— FRANK A H AND MOTT L O   1946 Studies of vesicular stomatitis with special reference to a virus of swine or pig Jour Amer Vet Med Assn 108 5

SIGURDSSON B   1943 The influence of host and temperature incubation on infection of the chick embryo with vesicular stomatitis virus Jour Exper Med 78 17

SKINNER H H   1951 Infect on of chickens and chick embryos with the viruses of foot and mouth disease and of vesicular stomatitis Nature (London) 174 1052

SORENSEN D K   1953 Virus infectivity transmission and immunity studies of vesicular stomat tis in cattle and sheep Doctoral thesis Univ of Wisc

STROZZI P AND RAMOS SACO T   1953 Teat vesicles as primary and almost exclusive lesions in an extensive outbreak of vesicular stomatitis (N J strain) in milking cows Jour Amer Vet Med Assn 123 415

THEILER, S   1901 Eine contagiose Stomatitis des Pferdes in Sud Afrika Deutsch tierärztl Wochenschr 9 131

TIMM E V   1955 The study of virus interaction by means of serological and tissue culture methods Doctoral thesis Univ of Wisc

WAGENER K   1932 Foot and mouth disease and vesicular stomatitis Jour Amer Vet Med Assn 80 59

mune animals would simplify diagnosis and control of other vesicular disease, but there is insufficient justification for it on the usual economic grounds While vesicular stomatitis in swine ready for market could be costly to the livestock owner in delay or shrinkage, under most conditions he is not concerned as the animals return to normal appearance and weight within 2 weeks

REFERENCES

ADAMS E, FERRIS, D, HANSON, R. P, AND KARSTAD, L. 1956 Annual report to U.S.D.A. on epi zootiology of vesicular stomatitis of swine by Department of Veterinary Science University of Wisconsin Mimeo

BACHRACH H L., CALLIS, J J, AND HESS W R 1955 The growth and cytopathogenicity of vesic ular stomatitis virus in tissue culture Jour Immunol 75 186

BANKOWSKI R A, AND KUMMER, M B 1905 Vesicular stomatitis and vesicular exanthema differ entiation by complement fixation Amer Jour Vet. Res 16 374

BAUDOU, A C 1939 Observaciones sobre estomatitis vesicular de los caballos Rev Med Vet. 21 361

BAUER J H, AND COX, H R 1935 Ultrafiltration of the virus of vesicular stomatitis Proc. Soc. Exper Biol and Med 32 567

BRADISH, C J BROOKSBY, J B AND DILLON, J F JR. 1906 Biophysical studies of the virus system of vesicular stomatitis Jour Gen Microbiol 14 290–314

BRANDLY, C A., CHOW, FUHO CHOW, T L., HANSON, R P, KOWALCZYK, T, LAZANO, E., AND SOREN SEN, D K 1930-53 Quarterly reports to Camp Detrick on vesicular stomatitis by the De partment of Veterinary Science University of Wisconsin Mimeo

——— HANSON R P, AND CHOW, T L 1951 Vesicular stomatitis with particular reference to the 1949 Wisconsin epizootic Proc Book 88th Ann Meet Amer Vet Med Assn, p 61

BROOKSBY, J B 1948 Vesicular stomatitis and foot and mouth disease differentiation by comple ment fixation Proc. Soc. Exper Biol and Med 67 254

——— 1949 Differential diagnosis of vesicular stomatitis and foot and mouth disease Exami nation of samples from Mexico with special reference to complement fixation Amer Jour Hyg 47 384

BURNET F M., AND GALLOWAY, I A 1934 The propagation of the virus of vesicular stomatitis in the chorioallantoic membranes of the developing hen s egg Brit Jour Exper Path 15 105

CAMARGO N C 1954 A contribution to the study of vesicular stomatitis in Mexico U S Live stock San Assn 58 379

CARREL A OLITSKY P K AND LONG P H 1928 Multiplication du virus de la stomatite vesi culaire du cheval dans les cultures de tissus Compt rend Soc. Biol (Paris) 98 827

CHOW, T L CHOW, FUHO, AND HANSON, R P 1954 Morphology of vesicular stomatitis virus. Jour Bact 68 724

——— and McNUTT, S H 1953 Pathological changes of experimental vesicular stomatitis of swine. Amer Jour Vet Res 14 420

COTTON W E 1926 The causal agent of vesicular stomatitis proved to be a filter passing virus Jour Amer Vet Med Assn 70 168

——— 1927 Vesicular stomatitis Vet Med. 22 169

COX, H R AND OLITSKY P K 1933 Neurotropism of vesicular stomatitis virus Proc. Soc. Exper Biol and Med 30 653

ELFORD W J AND GALLOWAY, I A 1937 Centrifugation studies III The viruses of foot and mouth disease and vesicular stomatitis Brit Jour Exper Path 18 155

FELLOWES O N, DIMOPOULLOS G T, AND CALLIS J J 1955 Isolation of vesicular stomatitis virus from an infected laboratory worker Amer Jour Vet Res 16 623

——— TESSLER, J HESS, W R VARDAMAN, T H., AND CALLIS J J 1956 Comparative titrations of vesicular stomatitis virus in various animal species and in tissue culture Amer Jour Vet Res 27 799-802

FERRIS, D HANSON, R P, DICKE, R J AND ROBERTS R H 1955 Experimental transmission of vesicular stomatitis virus by diptera Jour Infect Dis 96 181

FONG J, AND MARIN, S H 1951 Stability of vesicular stomatitis virus at varying S ion concen tration Proc Soc Exper Biol and Med 86 676

GALLO, P., RODIL, CALDERÓN, I A AND LUGO, L 1950 Identificación del virus de la estomatitis vesiculosa en Venezuela Rev Med Vet Parasit., Caracas 9 5

GALLOWAY, I A 1953 Personal communication

——— AND ELFORD, W J 1935 The differentiation of the virus of vesicular stomatitis from the virus of foot and mouth disease by filtration Brit Jour Exper Path 14 400

GRIFFIN T P, HANSON, R P., AND BRANDLY, C A. 1951 The effect of environmental temperature on susceptibility of the mouse to vesicular stomatitis viruses Proc Book, 91st Ann Meet Amer Vet Med Assn p 192

HANSON, R P 1952 The natural history of vesicular stomatitis virus Bact Res 16 179

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CHAPTER 12

Foot-and-Mouth Disease of Swine

Foot and mouth disease (FMD), known also as aphthous fever, epizootic aphthae, *fievre aphteuse* (French), *Maul und Klau enseuche* (German), *fiebre aftosa* (Span ish), and *afta epizootica* (Italian), is an acute, highly communicable disease affect ing almost exclusively cloven footed ani mals, domesticated and wild It is charac terized by the formation of vesicles in the mucosa of the mouth (tongue, lips cheeks gums, palate, etc), the skin especially on the snout, between and above the hoofs of the feet, the dew claws, teats, and udder

Confusion exists regarding the early his tory of FMD (Trautwein, 1929) The manifestations of the disease in the mouth, feet, udder, and other organs were con sidered as lesions and symptoms of several diseases, rather than one A century ago livestock owners, growers, and observers of livestock diseases did not recognize its importance since the infection generally causes comparatively low mortality, pro duces severe symptoms for only a relatively short time, and occurs in man with ex treme rarity It was not until the latter part of the nineteenth century and early in the twentieth century that the full importance and economic impact of FMD received proper consideration This re sulted in formation of commissions and establishment of laboratories in several countries for study of the disease

GEOGRAPHIC DISTRIBUTION

FMD occurs and generally is considered enzootic or frequently epizootic in most of the major livestock producing countries of the world, except in North America Central America, Australia and New Zea land In Australia FMD last occurred in 1872 (Seddon, 1953) Africa Asia Europe and South America have not been free from it for decades The British Isles have been free of the disease for months at a time only to have the disease reintroduced from European countries or other countries where the disease is enzootic The United States has experienced nine outbreaks of FMD (Mohler, 1938 Mohler and Traum 1912), the first in 1870 and the latest in 1929 In all but two instances the disease was eradicated and quarantines were re moved within a few months In the na tionally widespread outbreak of 1914–16 and in the 1924-25 outbreak in California 20 months of ceaseless effort was required before it was considered safe to remove all restrictions from the involved areas In the 1914–16 outbreak there were 35 092 swine, 77,210 cattle and 9 890 sheep and goats involved During the 1924-25 Cali fornia outbreak there were 21,195 swine, 58 791 cattle, 22 214 deer, and 29 775 sheep and goats destroyed because of infection or exposure to the disease Of the 22 214 deer destroyed 2,279 were found to be affected

bryonating chicken eggs, although generally regarded as non susceptible, have nevertheless been infected with some strains of virus passed intermittently through guinea pigs and incubated eggs (Traub and Schneider, 1948), or from 1 day old chicks to incubated eggs (Skinner, 1954, Gillespie, 1955) Hamsters have been shown by Korn (1953) and Komarov (1954) to be highly susceptible to plantar inoculation of FMDV, developing a severe and frequently fatal disease

Kidney Cell Cultures

Sellers (1955) and Bachrach *et al* (1955) reported that the virus of foot and mouth disease produced cytopathic changes in swine and bovine kidney cell cultures More recently, Bachrach *et al* (1957a) succeeded in propagating foot and mouth disease virus in cultures prepared from lamb kidneys, mammary carcinomatous tissue of mice, and in subcultures of bovine skin, muscle, and kidney Meléndez and co workers (1956) have reported success with cultures of embryonic bovine lung and heart tissue Methods have also been developed for the assay of FMDV and its antibodies as well as for the production of virus on a relatively large scale (Bachrach *et al*, 1955, Bachrach *et al*, 1957a)

ETIOLOGY

FMD is caused by a filtrable virus (Loeffler and Frosch, 1897) with an estimated particle size of 8–12 mμ as determined by ultrafiltration (Galloway and Elford, 1933) Seven immunologically and serologically distinct types of FMD have been reported types O and A by Vallée and Carré (1922), type C by Waldmann and Trautwein (1926), types SAT 1, SAT 2, and SAT 3 by the British workers at Pirbright, England (British Committee Report, 1954), and recently these same workers have reported an Asian type (Asia 1) (British Annual Report, 1951–55) Types O, A, and C have been reported from various parts of the world while the African types SAT-1, 2, and 3, have not been found outside of Africa Not much is

known at present regarding distribution of type Asia 1 except that it has been found in Pakistan, India, Hong Kong, and Thailand Immunological subtypes or variants of types O, A, and C, especially A, have been encountered frequently, and variants of SAT 1, 2, and 3 have also been reported (Henderson, 1954) The serological and immunological characteristics of the variants frequently are sufficiently different to cause difficulty in classification and immunization The resistance of the virus to physical and chemical influences is reported under Epizootiology (page 210) and Control (page 213)

CLINICAL SIGNS

The disease (Trautwein, 1929 Mohler 1938 Waldmann and Nagel, 1939 Mohler and Traum, 1942) manifests itself by the formation of vesicles on the mucosa of the mouth including the tongue, lips gums, pharynx and palate Vesicles may be found on the coronary band and on the skin between and above the hoofs Vesicles may be present on the teats of the nursing animals, and are frequently found on the snout and back of the rim of the snout and may sometimes extend into the nares In rare instances lesions may be found on other portions of the skin such as the perineum, vulva, or scrotum Lesions may be located at any one, several, or most of these sites

The vesicles are characteristic of FMD but are clinically indistinguishable in individual animals or groups of animals from lesions found in vesicular stomatitis (VS) or vesicular exanthema (VE) of swine (Fig 12 1) The vesicles usually rupture soon after their appearance, leaving raw, hemorrhagic, granular, eroded surfaces with ragged fragments of partially detached, more or less necrotic epithelium In the absence of secondary bacterial infections, the lesions tend to heal rapidly, beginning with a serofibrinous exudate and a gradual replacement of epithelium that may be unpigmented

The incubation period following natural exposure varies from 2 days to a week but

principally with foot lesions (U.S.D.A. Circular, 1926). In the smaller 1929 outbreak in California there were 3,291 swine and 277 cattle involved (Mohler, 1929).

The first diagnosis of FMD in Canada was in February, 1952 (Childs, 1952). The country was listed as freed from the infection by the U.S.D.A. on March 1, 1953. The presence of the disease in Mexico was established in 1946 (U.S.D.A. Release, 1947), and that country was not declared free until September, 1952, (Shahan, 1954) after nearly one million animals had been slaughtered and approximately sixty million vaccinations had been applied. The infection was again discovered in May of 1953 (U.S.D.A. Release, 1953) and restrictions against the importation of cattle, swine, sheep, and goats from Mexico into the United States were not again removed until December 31, 1954 (U.S.D.A. Release, 1954a, 1954b).

NATURAL AND EXPERIMENTAL HOSTS

Susceptibility to natural infection with *FMD* is primarily and almost exclusively limited to cloven footed animals, domestic and wild. Cattle, hogs, sheep, and goats are most frequently affected, in the order mentioned. However, this order of susceptibility to the disease does not always prevail. Adaptation of the virus to one particular species sometimes occurs. In such cases, pathogenicity for other normally highly susceptible species may be reduced, even to the point of apparently complete innocuity until the virus is again readapted to those species (Trautwein, 1929; Mohler, 1938). Outbreaks of FMD have been reported where swine were almost exclusively involved, although cattle and other susceptible animals were intimately exposed. The virus from two such outbreaks was shown under experimental tests to have developed a strong adaptation to swine, with high infectivity for this species, while cattle could only be infected experimentally with great difficulty (Waldmann, 1930; Brooksby, 1950). The reverse has also been observed. British Annual Report (1951-55) shows that foot-and-mouth dis-

ease virus (FMDV) isolated from cattle and known to be highly communicable and invasive for this species, displayed a low grade of invasiveness and communicability when tested in swine.

Dogs and cats, especially young ones, are slightly susceptible to artificial infection (Trautwein, 1929; Waldmann and Nagel, 1939; British Progress Reports, 1927a, 1931a, 1937a; Hove, 1929, 1930). Rats have been experimentally infected, and rare cases of natural infections of rats have been reported in England (British Progress Reports, 1931a). Rabbits have been infected artificially (British Progress Reports, 1927b, 1931b). The European hedgehog may be readily infected experimentally with FMD, and natural infections in hedgehogs have been observed in England (British Progress Reports, 1927b, 1931c, 1937b; Beattie *et al.*, 1928; McLauchlan and Henderson, 1947).

Olitsky *et al.* (1928) did not succeed in inducing lesions with either A or O type of foot-and-mouth disease viruses in 6 horses. Waldmann and Trautwein, reported by Trautwein (1929), could not produce the disease in 10 horses with the 3 types of FMDV known then. Trautwein (1929) also reports that Vallée was not successful in transmitting types A and O FMDV to horses.

Guinea pigs have been used extensively in FMD research since early 1920 (Waldmann and Pape, 1920, 1921; Olitsky *et al.*, 1928). The disease produced experimentally in this species is a prototype of the disease in cattle, but infection is not acquired by contact. Adaptation of the virus to guinea pigs sometimes requires repeated passages over a considerable period (British Progress Report, 1931d; Gins and Krause, 1921). Man is rarely infected with FMD, although in many countries he is extensively and repeatedly exposed to the disease, both directly and indirectly (Traum, 1951; Vetterlein, 1951). Suckling white mice have been found to be highly susceptible to intra abdominal injection of the virus, which causes spastic paralysis, myositis, and death (Skinner, 1951). Em-

but his observation has not been confirmed by other workers (Trautwein, 1925). Further study is needed to establish the character and even the presence of abnormal intranuclear or intracytoplasmic bodies in FMD lesions.

In some outbreaks and in individual animals, especially the young, there is parenchymatous degeneration and necrosis of the myocardium (Fig. 12.2) as manifested by discrete or confluent, gray-white or yellowish areas described as "tiger heart" (Bergman, 1913; Joest, 1911).

In inoculated, unweaned mice (Skinner, 1953), day-old chicks (Skinner, 1954; Gillespie, 1954), and in chicken embryos after adaptation of the virus, muscle tissue is especially affected (Traub and Schneider, 1948). Potel and Korn (1954) have shown

FIG. 12 2—Necrotic foci in pig heart caused by virus of foot-and-mouth disease, commonly referred to as "tiger heart." (Courtesy Drs. H. S. Frenkel and H. J. J. Fredericks, State Institute for Veterinary Research, Amsterdam, Netherlands)

that guinea pigs, after plantar intradermal inoculation of FMDV will, in many cases, develop myositis in the form of Zenkers' hyaline degeneration with mesenchymal cell infiltration of certain groups of skeletal muscles which contain virus of rather high titer. From these studies, these investigators are inclined to conclude that the virus multiplies in the muscle tissue.

DIAGNOSIS

The typical vesicle with blanched covering, with usually clear, sometimes turbid, colorless or straw colored fluid, is characteristic of FMD as well as VS or VE, and evidence of its existence is essential in clinical diagnosis of FMD or either of the other two vesicular diseases.

Field Diagnosis

Field diagnosis is usually accomplished by different types of inoculation of horses, cattle, and swine (Table 12.1) and, if other species such as sheep or goats are involved, these are included in the tests (Traum, 1934). Specially trained federal veterinarians are available in all parts of the United States, and field diagnosis should be left to them. When laboratory assistance is needed, diagnostic material will be referred to the laboratory by the diagnostician. In making a differential diagnosis the following criteria are considered:

1. Horses are not susceptible to FMD (Olitsky et al., 1928) but are susceptible to VS (Traum, 1934), and only slightly or irregularly so to VE (Madin and Traum, 1953; Traum, 1934). Natural infection of FMD in horses has not been proven.

2. Cattle are susceptible to FMD and VS (Cotton, 1927; Traum, 1934) but not to VE (Madin and Traum, 1953; Traum, 1934; Olitsky et al., 1928).

3. Swine are susceptible to all 3 diseases (Traum, 1934), and only swine have been naturally or experimentally infected with VE (Madin and Traum, 1953; Traum, 1934), except as noted above in 1.

In addition to VE and VS, swinepox may be suggestive of FMD. In swinepox the very superficial vesicles that may be

may be longer, depending on the particular strain of virus and upon the nature and extent of the exposure to infection. Experimentally inoculated animals usually develop clinical signs and lesions within 20 to 48 hours, but these may be delayed a week or more.

The lesions induce a series of other signs — increase in body temperature, lassitude, anorexia, excessive salivation, smacking of lips, and, when the feet are involved, lameness is evident, the severity depending upon extent of involvement. Because mastication may be painful and feed consumption is limited, due to anorexia, there is loss of weight and condition. There is a reduction or total cessation of milk flow. Abortion, mastitis, and chronic deformities of the feet are common. The mortality seldom exceeds 5 per cent. The greater mortality is in young animals and, in outbreaks where the heart is involved, the over-all mortality may be as high as 50 per cent.

PATHOLOGICAL CHANGES

In FMD the virus, naturally or experimentally introduced at susceptible sites such as the mucosa of the mouth or hair-

FIG. 12.1—Vesicles in coronary band typical of those found in foot-and-mouth disease, vesicular exanthema, or vesicular stomatitis.

less portions of the skin of the snout or feet, invades these parts, inducing vesicles From these primary lesions virus enters the blood stream and is distributed to various organs and tissues of the body. When it reaches sites of predilection, it usually sets up secondary vesicles with a rise in body temperature completing the typical diphasic clinical picture of vesicular diseases (Mohler and Traum, 1942; Trautwein, 1929). The formation of vesicles at secondary sites is influenced to a great extent by pressure in those areas; in heavy animals or animals trodding rough ground, there is apparently greater tendency toward involvement of the feet than in animals of lighter weight or those on well-bedded surfaces (British Progress Report, 1928a). It has been observed that when foot-and-mouth disease develops on farms or ranches where swine, especially the young, receive their feed from V-shaped troughs with cross bars on top, a high percentage of them show vesicle formation back of the rim of the snout. As found in the natural disease in the field, the vesicles may be either primary or secondary, and, in some instances, the point of entry may be situated where the primary lesion cannot be observed readily. The virus may enter naturally or be introduced experimentally at sites where primary vesicles do not develop. For example, in intramuscular injections the virus finds its way into the circulatory system and then is transported to sites of predilection where it forms vesicles. Teat and udder lesions may be primary as the result of nursing the infected young, or they may be secondary.

HISTOPATHOLOGY

Epithelial cells of the affected tissues are swollen and rounded with pycnotic nuclei. Polymorphonuclear cell infiltration, cell necrosis, separation of epithelial cells and layers, and subepithelial hyperemia are evident. The stratum spinosum and the stratum granulosum are especially involved (Trautwein, 1929; Galloway and Nicolau, 1928). Intranuclear inclusion bodies have been described by Huntemüller (1911).

3 Virus neutralization test With the advent of the use of the guinea pig as a suitable experimental animal for foot and mouth disease research by Waldmann and Pape (1920), the detection of immune bodies in recovered and hyperimmune animals was made practical (Olitsky et al, 1928, Waldmann and Pape 1921 Brooksby, 1949) These tests have used (1) in identification of antibodies in recovered animals or (2) in identification of virus In the first instance, serum from convalescent or recovered animals is tested for the presence of virus neutralizing antibodies such antibodies inhibit the growth of FMDV in tissue cultures (Sellers 1955 Bachrach et al, 1955) or reduce infectivity when measured mixtures of serum and virus are injected into susceptible animals Mostly, suckling mice and guinea pigs have been used for this purpose Suckling mice have given the better results (Skinner, 1953) Neutralizing substances are generally present by the end of the first week after appearance of clinical signs, or even sooner, and may be detectable in the serum several months after onset of the disease

In virus identification, the unknown agent is added to specific immune or hyperimmune serum of known neutralizing titer and inoculated into susceptible animals, such as cattle, guinea pigs, or mice Tissue cultures also may be used for this purpose (Sellers, 1955, Bachrach et al, 1955) The test has proved to be very satisfactory

4 Cross immunity test. This is one of the oldest tests and is still considered very reliable The difficulty has been to maintain sufficient animals immune to the various types of FMD, VS, and VE Identification of a virus is based on the relative resistance of known immune animals to the virus to be classified Immune guinea pigs and naturally susceptible immune animals are used for this test (Olitsky et al, 1928)

5 Guinea pig protection test. This is similar to the serum neutralization test except that the serum and virus are not mixed before injection into the guinea pig instead the serum is injected from a few minutes to one hour before injection of the virus Interpretation of the test is based on the presence or absence of secondary lesions with or without development of primary lesions In such a test it is important that the virus is known to produce regularly secondary lesions in guinea pigs (Olitsky et al, 1928 Brooksby, 1949)

6 Mouse inoculation test In the laboratory and under some circumstances in the field adult and suckling mice may aid in the differentiation of the three vesicular diseases, on the basis of the following facts VS virus quite regularly infects adult mice when inoculated intracerebrally, while FMDV, especially field virus is usually innocuous to adult mice by any route (Henderson, 1948, Nagel 1951 Cunha et al, 1955) FMDV, even that directly from field cases, (Skinner, 1951 1953) as well as vesicular stomatitis virus (VSV) (Fellowes et al, 1956) is highly pathogenic for unweaned mice Vesicular exanthema virus (VEV) appears to be innocuous for either unweaned or adult mice (Madin and Traum 1953)

7 Chicken inoculation test. Skinner (1954) reported that adult chickens were susceptible to the virus of FMD and VS when injected intradermally in the tongue producing vesiculation within 24 hours Similar results have been obtained with both of these viruses at the Plum Island Animal Disease Laboratory (PIADL 1955–57) Holbrook and Patterson (1957) have shown that adult chickens are not susceptible to the virus of vesicular exanthema when similarly injected

IMMUNITY

Recovery from natural or artificially induced FMD is followed by type specific and, to a degree, variant specific immunity (Trautwein, 1929, Olitsky et al, 1928 Waldmann and Nagel, 1939 British Progress Report, 1927c, Galloway, 1954, Mohlmann, 1954) Immunity in FMD and other vesicular diseases is classified as local or histogenic, and general or humoral Local immunity develops within 3 days at and around the site of the lesion Humoral immunity is detectable within a week,

found anywhere on the body are preceded by papules and followed by pustules (Creech, 1942), conditions which do not occur in FMD, VE, or VS Lameness due to traumata but without vesiculation occurs frequently in garbage fed swine and may be confused with FMD Sudden deaths suspected as being due to poisoning may be caused by the heart lesions of FMD

Laboratory Diagnosis

1 Complement fixation test C i u c a (1929) reported the demonstration of the presence of complement fixing antibodies in the sera of guinea pigs which had been infected or hyperimmunized with foot and mouth disease virus This test is used for the identification of virus in tissue (usually vesicle covering) from suspected cases of infection in the field (Traub and Mohlmann, 1943, Brooksby, 1952) In order to identify specifically the virus, the specimens must be fresh, field material is best preserved in buffered glycerin solution kept at 4° to 7° C, or it may be frozen Tissue from the lesions is used as antigen in the presence of specific hyperimmune serum of

the various vesicular diseases Guinea pig serum is most commonly used Marucci (1957) has devised a direct complement fixation test for detection of antibodies in sera from convalescing and vaccinated cattle

2 Indirect complement fixation test (Rice and Boulanger, 1953, Rice and Brooksby, 1953) This test is based on the observation that serum from convalescent or recovered cattle in the great majority of cases of FMD and VS does not sufficiently fix the complement in an ordinary complement fixation test to afford a diagnosis however, such serum may partially or totally engage known titrated antigen to the extent that the antigen will no longer have the power to show the maximum fixation nor possibly any fixation in the presence of known, titered, hyperimmune serum Under some circumstances, the test has been found to have definite practical application in FMD and VS in cattle and it is suggested for use with swine sera (Rice et al, 1953) This test has not been used to any great extent instead the serum neutralization test is used whenever possible

TABLE 12 1

RESPONSE IN ANIMALS TO INOCULATION BY VARIOUS ROUTES WITH THE VESICULAR VIRUSES *

Test Species	Route of Inoculation	Minimal Number of Animals Needed	Typical Response If Unknown Virus Is		
			FMD	VS	VE
Swine	intradermal (snout and inner lips, plus scarified snout)	2	+	+	+
	intravenous	2	+	+	+
Horse	intramuscular	1	−	+	+
	intradermolingual	1	−	+	⇌
Cattle	intradermolingual	1	+	+	−
	intramuscular	1	+	− †	−
Sheep	intradermolingual	2	+	⇌	−
Guinea pigs	intradermal (plantar pads)	2	+	+	−
Suckling mice	intraperitoneal	10	+	+	+
Adult mice	intracerebral	10	−	+	−
Embryonating chicken eggs	allantoic cavity	5	−	+	+
Adult chickens	subcutaneously in the tongue	5	−	+	+

* Modified from Madin and Traum (1953)
Key to symbols
+ = positive
− = negative
⇌ = irregular and slight.
† With rare exceptions

survived best in distilled water when the acid base reaction was adjusted to pH 7 6 Conditions other than pH of the medium may influence survival time of the virus

During a 5 week period of observation Bachrach et al, (1957a) found only a slight decrease in infectivity of FMDV produced in tissue cultures in veronal acetate buffer solution at 4° C and pH 7 or 7 5 At pH 8, 90 per cent of the virus infectivity was lost in 3 weeks, at pH 9, a corresponding loss occurred in 1 week In samples stored at pH 6 5 and 10, there was a 90 per cent reduction in infectivity every 14 hours and, in samples at pH 5 and 6, a similar reduction was observed in less than 1 minute Under conditions of these experiments the rates of inactivation of virus in 3 solutions at pH 2, 3, and 4 were too rapid to be measured

Other Influences on Virus Stability

Outside the animal body variable conditions affect virus viability (Trautwein, 1929) When exposed to sunlight, especially in a thin layer, the virus is readily destroyed, but in tissue fragments containing the virus, or on contaminated materials such as hair, feed, and stable equipment the virus may remain infective for several weeks under average stable and farm conditions (British Progress Reports, 1927f, 1928b)

In open sewage, Wagener (1928) found the virus to persist for 39 days and, under certain conditions as long as 103 days, but when the infected sewage was enclosed under conditions permitting concentration of ammonia, the virus was destroyed within 2 days

That the virus does not always perish quickly outside the animal body is strongly indicated by the fact that FMD has in rare instances appeared on premises during the gradual restocking which is usually started, under the eradication policy in the United States, 30 to 60 days after slaughter of affected and contact animals and disinfection of the premises (Trautwein, 1929, Waldmann and Nagel, 1939) Evidence is available to show that, in one instance in

California the virus persisted on such premises for 345 days (Traum 1934 Mohler and Traum 1942)

It has been accepted generally that heat sufficient to destroy non spore bearing bacteria will inactivate FMDV, and that pasteurization temperatures of 142 145° F (61–63° C) for 30 minutes are adequate to destroy FMDV, providing every portion of the medium containing the virus is maintained at that level Further, it has been accepted generally that virus kept at a temperature of 37° C loses its infectivity in a few days At room temperature it remains viable somewhat longer, but under ordinary refrigeration (4–7° C) it may remain infective for many months At low temperatures (30 to 70° C) the virus remains viable for several years (British Progress Report, 1927d)

It now appears from unpublished results of work done at the Plum Island Animal Disease Laboratory (1957) that whereas FMDV contained in bovine tongue lesions subjected to pasteurization temperatures for 30 minutes loses 90 to 99 per cent of its infectivity, some residual infectivity may be demonstrated under certain conditions

Season and Climate

Apparently season and climate have no effect on the spread of FMD except as they may affect traffic and movement of animals and other agricultural products, as well as people Possibly hot, dry weather may tend to slow epizootics

Chemical Disinfectants

The effects of various chemical disinfectants on the virus of FMD have received considerable attention in the past (Trautwein, 1929, Olitsky et al, 1928, British Progress Report, 1927d) In general, it may be stated that most of such materials have for various reasons been proved unsatisfactory for practical use An exception to this general rule is sodium hydroxide (NaOH, caustic soda lye) (Olitsky et al 1928 British Progress Report 1928c) A

usually after 5 days, the peak being reached in 3 to 4 weeks Local immunity to direct inoculation with FMD usually endures for 3 to 4 months, decreasing rapidly there after, although resistance to infection by contact may persist as long as 2 years Humoral immunity against FMD may persist for 2 or 3 years but generally begins to subside materially at the end of 1 year Immunity appears to be less substantial and persistent in swine than in cattle (British Progress Report, 1937c)

TREATMENT

In those countries where the slaughter or the stamping out method of eradication is used there is no place for treatment There is no known specific cure for the disease, and the palliative treatment only alleviates the symptoms of the disease and does not prevent the spread of infection The repeated handling of animals in attempts at treatment interferes with the eradication effort

EPIZOOTIOLOGY

In epizootiology and control, it is important to have a good understanding of sources of infection, transmission, communicability, and resistance of the virus to chemical and physical influences

Source of Infection

The virus of FMD is present in the fluid and coverings of the vesicles and may be found in the blood, organs, secretions, and excretions in the febrile stages of infection The conditions under which the virus occurs in and out of living and slaughtered animals determine its viability and infectivity In the living animal, virus in the vesicle coverings and fluid, in other portions involved in the vesicles, and in most of the body tissues and organs, loses its infectivity within 5 to 7 days after appearance of lesions, with infrequent exceptions It has been found that certain portions of the living animal, such as the skin, hair, and loose portions and crevices in the hoofs may, in some cases, contain infective virus for some time (Mohler,

1938, Trautwein, 1929, Waldmann and Nagel, 1939)

Effect of pH on Virus Stability

In the slaughtered animal (Trautwein 1929, Stockman and Minett, 1926) the formation of sarcolactic acid in the normal process of rigor mortis rapidly inactivates virus that may be contained in the muscles and other parts reached by the acid Quick freezing, however, suspends acid formation, and such muscle may retain its infectivity until thawed Lymph nodes, liver, kidney, bone marrow, rumen (of cattle), and other organs, as well as residual blood are not affected by the changes attending rigor mortis and may retain infective virus for weeks, depending on circumstances Henderson and Brooksby (1948), who contributed substantially to this knowledge, based their conclusions primarily on studies with infected bovine tissues, and in all but one experiment the infectivity of the materials was tested in cattle In one case, however, bovine material was fed to hogs Henderson and Brooksby state, 'We have made no observations on the survival of virus in porcine tissue, but pH studies showed that the acid formation of rigor mortis was not sufficiently different from that of beef and beef offal to necessitate separate consideration '

The literature on FMD contains many references on the effects of the hydrogen ion concentration on stability of FMDV In general, it has been found that media at pH 7 4–7 6 are optimal A shift in pH in either direction makes conditions for survival less favorable, but not materially so until the pH on the acid side is below 6 and on the alkaline side is above 9 Olitsky and Boez (1927), studying conditions most suitable for multiplication of FMDV, state that the hydrogen ion concentration of the medium should be 7.5 to 7 6

Henderson and Brooksby (1918) found in their experiments that the pH of meat did not go below 5 3 and that at this pH there was destruction of the virus

Edwards (1931) found that the virus

attributed to swill and 36 were in herds with possible contact with swill

Communicability

Affected animals are most infective during the acute stages of the disease (Mohler, 1938, Trautwein, 1929, British Progress Report, 1927e) There is appreciable field evidence to indicate that some animals continue to carry the virus for indefinite periods after recovery (Waldmann *et al*, 1931, Traum, 1934, Fluckiger, 1943)

Generally, all animals of a susceptible species in an exposed herd develop infection in time, but, under some circumstances, the rate of incidence is considerably less than 100 per cent The possibility of inapparent infections should be considered, since such cases have been reported

CONTROL

Preventive Measures

The exclusion of animals of susceptible species and fresh chilled or frozen meats therefrom, and restrictions on animal products originating from infected countries constitute the most effective preventive measures that may be taken, especially in countries like the United States that are largely self sufficient insofar as livestock production is concerned Included among these prohibitions is garbage containing meats from ships and aircraft originating in foreign countries where FMD exists Some products considered to be potentially dangerous, such as hides, and animal casings and glands, may be permitted entry into the United States but only under prescribed conditions whereby the products are processed under supervision at designated official establishments

Special Actions Applicable to Epizootics

In many countries, reporting of such diseases as FMD is mandatory, and failure to do so is punishable by fine or imprisonment Every livestockman and veterinarian, as well as others, must be held responsible for immediately reporting any illness in livestock suspected to be FMD Such reports are to be made to the state or federal disease control officials in the state

In addition to prompt imposition of effective quarantines and immediate establishment of inspection procedures for the purpose of checking all possible contact herds the prompt disposal of infected and exposed animals and thorough cleaning and disinfection of affected premises constitute the chief means of combating the disease

Up to the end of the first quarter of this century there were virtually only two important methods of control of FMD The first depended primarily on isolation quarantine, and disinfection the other included these, plus the slaughter of infected and exposed animals In the 1920's, the first method was supplemented in some European countries by the use of convalescent or hyperimmune serum In most European countries there has been legal authority for slaughter of infected and exposed animals if, in the opinion of those responsible for control of animal diseases such procedure is considered economically and otherwise practical and effective in each circumstance Slaughter has seldom been used however, in countries frequently subjected to epizootics, and with the introduction of FMD vaccines many countries depend largely on such products for control of the disease

Beginning with the 1902 outbreak, the United States has used the stamping out or slaughter method of eradication Great Britain, Eire Norway, Canada, South Africa, the Rhodesias, Australia, and New Zealand also rely upon the slaughter method to eradicate the disease After a two year study in the British Isles and many other countries, the Departmental Committee on Foot and Mouth Disease (British Committee Report, 1954) appointed by Great Britain's Minister of Agriculture and Fisheries, stated in its report

In the circumstances of today, and of the immediate future, so far as they are foreseeable any idea that it would be possible to do away with stamping-out by making the whole susceptible animal population—or even all cattle—immune by vaccination is in the realm of fan

1 per cent solution of NaOH at pH 13 4
destroys the virus in less than 1 minute In
practice, 2 per cent NaOH (pH 13 7) is
generally used, if less than 1 per cent is
employed, longer exposures are required
Sodium carbonate in 4 to 5 per cent solu
tions, depending on the number of mole
cules of water in the chemicals used, has
proved satisfactory where speed of action
is not urgent (British Progress Report,
1931e, Trautwein and Reppin, 1928)
Suspensions of infected bovine tongue epi
thelium have been inactivated by exposure
to ethylene oxide gas for a 4 hour period
(Callis et al, 1957) Many new virucidal
preparations are appearing on the market
their efficacy against FMDV remains to be
determined

Milk and Milk Products

In milk and milk products, viability of
FMDV depends generally on the degree
and rate of acid formation as well as
temperature (Trautwein, 1929, Waldmann
and Nagel, 1939, Terbruggen, 1932)
Thus, in fresh unpasteurized milk, at in
cubator temperature (37° C) the virus is
destroyed within about 24 hours, at room
temperature (18 to 20° C) in about 6
days, at refrigerator temperature (4 to 6°
C) in approximately 12 days On the
other hand, milk pasteurized or heated to
higher temperatures and then artificially
contaminated with virus may harbor viable
virus for 30 days at refrigerator tempera
ture Virus persists longer in cream than
in skimmed milk

In butter made from sour cream, FMDV
is rapidly destroyed In salted butter made
from sweet cream, it may remain viable
for 14 days, in unsalted butter, 8 days

The products of cheese manufacturing,
such as the whey and residual curds fre
quently fed to pigs, are dangerous since
they are usually used soon after they be
come available Most types of cheese, when
marketed, have undergone heating and
ripening processes with sufficient heat and
shift in pH to the acid side to cause in
activation of the virus

Modes of Transmission

Direct or indirect contact with infected
animals or carcasses and organs, excretions
and secretions of infected animals or con
tact with contaminated objects or animals,
especially man, may transmit infection to
susceptible animals Milk, creamery prod
ucts, sera and other biologic products,
pastures, barns, pens, stockyards, sale yards
feed, garbage, railway cars, and trucks are
included (Mohler, 1938, Trautwein, 1929,
Waldmann and Nagel, 1939, British Com
mittee Report, 1954)

All outbreaks in the United States prior
to 1902 were attributed to imports of cat
tle from infected countries (Mohler,
1938) The source of the 1902 outbreak
was traced to a case near the Chelsea, Mas
sachusetts, docks, it being suspected that
infection was introduced through foreign
shipments, either by hay, straw, halters,
ropes, hides, hair, or wool There was some
evidence, however, that the disease might
have been introduced through importa
tion of vaccinia virus contaminated with
FMDV, as was proved to be the case in
the 1908 epizootic (Mohler and Rosenau
1909) There were definite indications that
the 1914 outbreak, first discovered near
Niles, Michigan, began when hogs were
fed trimmings and offal from a packing
house which handled foreign meats (Moh
ler, 1924).

The outbreaks of 1924 and 1929 in Cali
fornia are attributed to hogs fed raw gar
bage from vessels carrying meat stores ob
tained in countries where FMD was en
zootic (Mohler, 1938) The source of in
fection in the Texas outbreaks of 1921 and
1925 is unknown (Mohler, 1938)

British authorities (British Committee
Report, 1951) attribute primary out
breaks from 1938 to 1953 to the following
probable sources feeding of swill (gar
bage), contact with imported meats and
bones (other than swill) and infected
serum, and there is strong circumstantial
evidence to indicate that migrations of star
lings from the continent of Europe may
have been contributory in some cases. Of
510 outbreaks during this period, 214 were

not as extensively vaccinated as cattle The vaccine most commonly used is that developed by Schmidt (1938) and Waldmann and Kobe (1938) (Waldmann and Nagel 1939, Galloway 1954 Mohlmann 1954, Camargo and Mott, 1953) This product consists essentially of aluminum hydroxide adsorbed virus inactivated by formaldehyde and incubation Variations of the Schmidt Waldmann vaccine have been used in various countries Generally virus for the vaccine is derived from the lingual epithelium of artificially inoculated cattle Frenkel (1951) has developed techniques for propagation of the virus in freshly explanted tongue epithelium from normal slaughter cattle It is generally considered that vaccine produced with bovine tongue epithelium infected with bovine adapted virus does not produce satisfactory immunity in swine (Fogedby and Frenkel 1947), however, when bovine adapted strains are adapted to hogs by repeated passages in this species and then injected into the tongue of susceptible cattle, such infected epithelium produces a vaccine which is more satisfactory for immunizing swine and also suitable for cattle immunization (Mohlmann, 1950)

Another type of vaccine consists of whole blood of viremic animals treated with crystal violet (Galloway, 1954), as is done in the production of C V hog cholera vaccine This type of FMDV vaccine has proved to be effective but somewhat impractical and expensive to produce except on a limited scale Several other types of vaccine have been proposed and used to a limited extent in certain localities (Belin and Belin 1949, Thomas et al, 1952)

In using vaccine, knowledge of the type of virus prevailing in the outbreak is essential In some areas, bivalent or trivalent vaccines have been used (Geiger, 1951) There is some question as to the efficacy of polyvalent vaccine, especially

when more than two types of virus are used (Michelsen, 1953) All vaccines should be critically tested to prove their innocuity as well as their potency

Sera

Passive immunity Hyperimmune serum or convalescent serum taken from animals in 2 to 4 weeks after onset of the disease, confers temporary passive immunity Such products were widely used in Europe in the middle and late 1920 s and are still used to some extent in South America As late as the 1937–38 outbreak of the disease in West Germany, there was a weekly production of 40 000 to 50 000 liters of hyperimmune serum (Loeffler and Uhlenhuth 1901 Ernst, 1923 Waldmann and Nagel 1939 Mohlmann, 1954)

Import Restrictions

In the United States import restrictions are formulated and administered by the Animal Inspection and Quarantine Division of the Agricultural Research Service USDA under the authority of Section 306 (a) of the Tariff Act of 1930 and the Acts of 1890 and 1903 The regulations are subject to revision from time to time in accordance with requirements of changing conditions

Generally, movements of people are restricted only in relation to quarantined areas and quarantined premises during an epizootic However, the baggage of all persons entering the United States from countries where FMD and other exotic infections are present is subject to close inspection

Public Health Aspects

There have been very few scientifically authenticated cases of infection with FMD virus in man (Traum, 1951 Vetterlein, 1954) The disease in man has never become a public health problem

REFERENCES

BACHRACH H L, BREESE, S S JR, CALLIS, J J, HESS, W R, AND PATTY, R E 1957a Inactivation of foot and mouth disease virus by pH and temperature changes and by formaldehyde Proc. Soc. Exper Biol and Med 95 147

——, CALLIS J J HESS, W R, AND PATTY, R E, 1957b A plaque assay for foot and mouth disease virus and kinetics of virus reproduction Virology 1(2) 224–30

tasy In present circumstances stamping out must continue to be the policy in Great Britain

Other countries such as West Germany, Belgium, France, Italy, Spain, and most of the South American countries depend largely upon vaccination, while Switzerland, Denmark, Holland, Sweden, and Finland, when considering it appropriate, make use of stamping-out methods as well as vaccination In all countries restrictions, quarantines, and sanitary measures are employed in varying extent.

Stamping-Out Method

Briefly, the stamping out method consists of

(1) Prompt slaughter and proper disposal of animals infected with, or exposed to, FMD removes at once the greatest source of active virus and avoids the possibility of carriers Slaughter and burial or incineration are carried out as rapidly after the establishment of a diagnosis as possible Under favorable circumstances in the United States, this has been accomplished within a few hours after discovery of infection Early discovery and diagnosis is paramount in prompt eradication of the disease

(2) Thorough cleaning and disinfection of the affected premises and of materials possibly contaminated with virus removes and destroys the greater portion of what ever active virus may remain after proper burial or burning of slaughtered animals

(3) Following an appropriate interval of time after disinfection of premises, test animals, including cattle and hogs, are placed to feed and graze and otherwise come in contact with all parts of the premises and objects which may have been contaminated with FMD virus Hogs are especially desirable because of their rooting habits Occurrence of the disease in test animals reveals any virus that may have survived the cleaning and disinfecting procedures The effectiveness of these procedures is shown by the records of some 5,000 premises cleaned and disinfected in out breaks in this country since 1902 (Traum, 1934) There were only 14 instances in the 1914–16 outbreak and only 2 in the Cali

fornia outbreak of 1924–25 where infection developed in test animals after cleaning and disinfection, or only 0 3 per cent. These were detected by the test animals before complete restocking had been per mitted (Mohler and Traum, 1942, Traum, 1934, British Committee Report, 1954)

(4) Authority for quarantine of affected premises rests primarily with the officials of the state, although affected areas or en tire states may be subjected to quarantine by the federal government

The area surrounding the infected prem ises within a distance of 5 to 50 miles or more, depending upon circumstances should be quarantined immediately, pend ing close inspection of all contact premises and elimination of the infection wherever found

(5) Complete isolation of infected premises, with prohibition of traffic there from pending disposal of animals, disin fection, and testing of the premises, is the first requirement in combating FMD

(6) Inspections are usually systematized and coordinated among federal, state, and other local authorities

(7) Cleaning and disinfection proce dures are carried out under direct official supervision of state and federal animal disease eradication and control personnel (U S D A , 1943)

(8) Indemnities provided by the federal and state governments are paid to owners of animals or property destroyed in the course of eradicating FMD

(9) Insofar as is known, FMD is not transmitted by insect vectors (British Pro gress Report, 1937a) , nor has there been evidence in the course of past epizootics in the United States to indicate that birds were an important factor in dissemination of the disease People, dogs, cats, farm and wild animals, and vehicles that may act, directly or indirectly, as mechanical trans mitters of the virus are subject to close control in quarantine areas

Vaccines

Active immunity. This is an important tool for control of the disease in many countries where it is enzootic, but hogs are

GEIGER, W 1954 Verification de la valeur du vaccin anti aphteux trivalent Bull Off int Epiz 41 1, 97

GILLESPIE, J N 1954 The propagation and effects of type A foot and mouth disease virus in day old chicks Cornell Vet 44 425

——— 1955 Propagation of type C foot and mouth disease virus in eggs and effects of the egg cultured virus on cattle Cornell Vet. 45 2 170

GINS, H A AND KRAUSE, C 1924 Zur Pathologie der Maul und Klauenseuche Ergeb allg Path Mensch u Tiere 20 805

HENDERSON, W M 1948 Some observations on the quantitative study of vesicular stomatitis virus Jour Comp Path and Therap 58 172

——— 1954 The Nature of Foot and Mouth Disease Rep Dept Comm on Foot and Mouth Disease 1952–54 Brit Min Agr and Fisheries London p 91

——— AND BROOKSBY, J B 1948 The survival of foot and mouth disease in meat and offal Jour Hyg 46 394

HOLBROOK, A A AND PATTERSON, W C 1957 A preliminary report The use of mature chickens in the differential diagnosis of vesicular exanthema and vesicular stomatitis Jour Amer Vet Med Assn 131 196–97

HOVE K R 1929 Die Maul und Klauenseuche bei Katzen Arch wiss prakt Tierheilk 60 123

——— 1930 Die Uebertragung der Maul und Klauenseuche auf Hund Arch wiss prakt Tier heilk 62 483

HUNTEMULLER, O 1911 Befunde bei Maul und Klauenseuche Zentralbl f Bakt I Abt Orig 61 375

JOEST, E 1911 Untersuchungen uber die Myokarditis bei bosartiger Aphthenseuche Zeitschr f Infektionskrankh , parasit Kronkh u Hyg d Haustiere 10 120

KOMAROV, A 1954 Propagation of foot and mouth disease virus in the Syrian hamster Refuah Vet 11 198

KORN, G 1953 Die Pathogenese und Histogenese der Maul und Klauenseuche des Goldhamsters Arch f Exper Veterinärmed. 7 192

LOEFFLER, F, AND FROSCH, P 1897 Summarischer Bericht uber die Ergebnisse der Untersuchungen der Kommission zur Erforschung der Maul und Klauenseuche Zentralbl f Bakt I Abt Orig 22 257, 23 371

——— AND UHLENHUTH, P 1901 Über die Schutzimpfung gegen die Maul und Klauenseuche im besondern uber die praktische Anwendung eines Schutzserums zur Bekampfung der Seuche bei Schweinen und Schafen Zentralbl f Bakt I Abt 29 19

McLAUCHLAN, J D , AND HENDERSON, W M 1947 The occurrence of foot and mouth disease in the hedgehog under natural conditions Jour Hyg 45 474

MADIN, S H , AND TRAUM, J 1953 Experimental studies with vesicular exanthema of swine Vet Med 48 395, 443

MARUCCI, A A 1957 Direct complement fixation for detection of foot and mouth disease anti body in serums from experimentally infected cattle Amer Jour Vet Res. Accepted for July publication

MELÉNDEZ, L V , GAGGERO, A C RODRIGUEZ, R T , and NORAMBUENO, M G 1956 Instituto Bacteriológico de Chile, Santiago, Chile Personal communication

MICHELSEN, E 1953 Expérimentation et résultats d expériences sur un vaccin anti aphteux tétravalent Bull Off int Epiz 38 1, 65

MOHLER, J R 1924 Foot and mouth disease with special reference to the outbreak of 1914 U S D A Dept Circ No 325 2

——— 1929 The 1929 outbreak of foot and mouth disease in California Jour Amer Vet Med Assn 75 309

——— 1938 Foot and mouth disease U S D A , Farmers Bull No 666, p 1 (Revised 1952)

——— AND ROSENAU, M J 1909 The origin of the recent outbreak of foot and mouth disease in the United States U S D A , Bur Anim Ind Circ No 147, p 1

———, AND TRAUM, J 1912 Foot and mouth disease Separate No 1882, Keeping Livestock Healthy, 1912, Yearbook of Agr U S D A.

MOHLMANN, H 1950 Die aktive Immunisierung des Schweines gegen Maul und Klauenseuche Arch f Exper Veterinärmed 2 79

——— 1954 Stand der Forschung über das Virus der Maul und Klauenseuche Arch f Exper Veterinärmed 8 316

NAGEL, H C 1954 Personal communication

OLITSKY, P K AND BOEZ, L. 1927 Studies on the physical and chemical properties of the virus of foot and mouth disease. IV Cultivation experiments Jour Exper Med 45 533

———, TRAUM, J AND SCHOENING, H W 1928 Rep of the Foot and Mouth Dis Comm U S D A Tech Bull No 76

LONG ISLAND ANIM DIS. LAB. 1957 Unpublished work

——— 1955 57 unpublished work

POTEL, K AND KONN, G 1954 Experimentelle Untersuchungen uber das Vorkommen von Skelet tmuskelveranderungen bei mit Maul und Klauenseuche infizierten Meerschweinchen. Arch f Exper Veterinärmed 8 1

BACHRACH, H L , HESS, W R AND CALLIS, J J 1955 Foot and Mouth disease virus Its growth and cytopathogenicity in tissue culture Science 122 3185, 1269

BEATTIE, J M , MARCOS, Z , AND PEDEN, D 1928 The transmission of foot and mouth disease in rodents by contact Jour Comp Path and Therap 41 353

BELIN, M AND BELIN, C 1949 Le vaccin antaphteux Belin dans l epizootie de la Sarre Bull Acad vét France 22 357

BERGMAN, A M 1913 Veranderungen in der Herzmuskulatur bei apoplektischen Fallen von Maul und Klauenseuche bei Ferkeln Zeitschr f Infektionskrankh , parasit Krankh u Hyg d Haustiere 14 422

BRITISH ANNUAL REPORT 1954–55 Foot and Mouth Disease Res Inst Pirbright, England, p 11

BRITISH COMMITTEE REPORT 1954 Rep Dept Comm on Foot and Mouth Disease, 1952–54 Brit Min of Agr and Fisheries, London, p 7

BRITISH FOOT AND MOUTH DISEASE COMMITTEE 1927a 2nd Prog Rep, p 50
——— 1927b 2nd Prog Rep p 17
——— 1927c 2nd Prog Rep p 80
——— 1927d 2nd Prog Rep pp 47, 70
——— 1927e 2nd Prog Rep p 15
——— 1927f 2nd Prog Rep p 70
——— 1928a 3rd Prog Rep p 96
——— 1928b 3rd Prog Rep p 81
——— 1928c 3rd Prog Rep p 27
——— 1931a 4th Prog Rep , p 66
——— 1931b 4th Prog Rep p 21
——— 1931c 4th Prog Rep p 164
——— 1931d 4th Prog Rep p 65
——— 1931e 4th Prog Rep p 57
——— 1937a 5th Prog Rep , p 29
——— 1937b 5th Prog Rep , p 56
——— 1937c 5th Prog Rep p 21

BROOKSBY, J B 1949 The antibodies in foot and mouth disease Agr Res Council Rep No 9 His Majesty s Stationery Office, London
——— 1950 Strain of the virus of foot and mouth disease showing natural adaptation to swine Brit Jour Hyg 47 184
——— 1952 The technique of complement fixation in foot and mouth disease research Agr Res Council Rep No 14, His Majesty s Stationery Office, London, p 1

CALLIS, J J , TESSLER, J , FELLOWES, O N , AND POPPENSIEK, G C 1957 Inactivation of foot and mouth disease virus by ethylene oxide gas Proc Soc Amer Bact , A17 22

CAMARGO, F , AND MOTT, L O 1953 The first year s production and testing of twelve million doses of foot and mouth disease vaccine by the Mexican United States Commission for Eradication of Foot and Mouth Disease Bull Off int Epiz 39 5/6, 435

CHILDS T 1952 The history of foot and mouth disease in Canada Proc 56th Ann Meeting U S Livestock San Assn p 153

CILEA, A 1929 The reaction of complement fixation in foot and mouth disease as a means of identifying the different types of virus Brit Jour Hyg , 28 325

COTTON, W E 1927 Vesicular stomatitis Vet Med , 22 169

CREECH, G T 1912 Swine pox Yearbook of Agr , U S D A , p 714

CUNHA, R G , LICHORS, E A , AND MATA, O 1955 Difference between foot and mouth disease and vesicular stomatitis viruses by means of mouse inoculation Amer Jour Vet Res 16 60, 472

EDWARDS, J T.. 1931 The respiratory exchange of FMDV — Its significance in determining the conditions required for the survival and growth Brit Foot and Mouth Dis. Comm 4th Prog Rep, p 170

ERNST, W 1925 Uber Wirkung und Prüfung von Maul und Klauenseucheserum Münch tierärztl Wschr 74 681

FELLOWES O N , DIMOPOLLOS, G T TESSLER, J., HESS, W R , VARDAMAN, I N AND CALLIS, J J 1956 Comparative titrations of vesicular stomatitis virus in various animal species and tissue cultures. Amer Jour Vet Res, 17 65 799

FLUCKIGER, G 1913 In welchem Ausmass sind von der Maul und Klauenseuche genesene Tiere Ansteckungsträger Zeitschr f Infektionskrankh, parasit Krankh u Hyg d Haustiere 59 220

FOGEDBY, E E, AND FRENKEL, H S 1947 La vaccination anti aphteuse (expériences danoises et néerlandaises). Bull Off int Epiz 28 p 5

FRENKEL, H S 1951 Research on foot and mouth disease III The cultivation of the virus in explantations of tongue epithelium of bovine animals Amer Jour Vet Res. 12 147–150
——— AND ELFORD, W J 1953 The determination of the virus of vesicular stomatitis from that of foot and mouth disease by filtration Brit Jour Exper Path 14 400.
——— AND NICOLAU, S 1953 Histological study of the development of foot and mouth disease in the tongue of the guinea pig rabbit and ferret Brit Foot and Mouth Dis. Comm 3rd Prog Rep, App III, p 101

GALLOWAY, I A.. 1951 Immunity to foot and mouth disease. Rep of Dept Comm on Foot and Mouth Disease Brit Min of Agr and Fisheries London, App XVI, p 118

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Pseudorabies (Aujeszky's Disease, Mad Itch, Infectious Bulbar Paralysis)

Pseudorabies is an acute infectious disease caused by a filtrable virus In cattle, cats and dogs, it is characterized by a marked local pruritis and an invariably fatal termination In swine there is no pruritis and the clinical severity of the disease is extremely variable Porcine pseudorabies is of veterinary importance from two rather different standpoints First the disease itself may be of serious importance in young pigs, though a very minor clinical ailment in older swine Secondly, swine appear to play a major role in the epidemiology of pseudorabies in cattle and, as will be discussed more fully later, older swine, subclinically infected with pseudorabies, are the probable source from which cattle acquire their fatal infections

HISTORY

Pseudorabies has occurred in the United States for many years and Hanson s (1954) account of the history of the disease furnishes an excellent record of its early prevalence here There is evidence that it prevailed at least as early as 1813 in Ohio The disease was referred to as mad itch' in the early American literature, and this designation has persisted up to the present time in popular parlance

Aujeszky (1902), working in Hungary, was the first to describe the disease in the scientific literature He observed its natural occurrence in cattle, cats, and dogs,

and transmitted the virus experimentally to rabbits He had initially suspected that the condition was rabies but the behavior of its causative agent in rabbits clearly differentiated it from this disease and Aujeszky, therefore, described it as a new infectious disease of domestic animals The resemblance of certain clinical aspects of the disease to those seen in rabies led to its designation as pseudorabies

Aujeszky made a thorough study of the causative agent of pseudorabies in experimentally infected rabbits guinea pigs and mice He observed that it was present not only in the central nervous system but in the blood as well, that it remained viable in glycerol for relatively long periods of time, and that it could be inactivated by phenol or heating He was unable to filter the agent through bacterial filters It remained for Schmiedhoffer (1910) and Sangiorgi (1914) to demonstrate the filtrability of the pseudorabies virus

Shope (1931) established that mad itch occurring in cattle in Iowa was the same as the disease that Aujeszky had described and that the mad itch virus was immunologically identical with Aujeszky s Hungarian strain of pseudorabies virus

ETIOLOGY

The virus which causes pseudorabies is about 100 mμ in diameter and readily filtrable through either Berkefeld or

Rice, C E and Boulanger P 1953 The use of direct and indirect complement fixation tests for the demonstration of antibodies for vesicular viruses in cattle Proc 90th Ann Meet Amer Vet. Med Assn, p 169

——— and Brooksby, J B 1953 *Studies of the complement fixation reaction in virus systems* V In foot and mouth disease using direct and indirect methods Jour Immun 71 300

——— ——— and Boulanger P 1953 The indirect complement fixation test in the detection of antibody in antiviral sera 6th Cong Internat di Microbiol 2 (Sec. 6-7) 196

Schmidt S 1938 Adsorption von Maul und Klauenseuchevirus an Aluminumhydroxyd unter besonderer Berucksichtigung der immunisierenden Eigenschaften der Virus adsorbate Zeitschr Immunitatsf 92 392

Seddon R 1953 Foot and Mouth disease, diseases of domestic animals in Australia Ser Pub Div Vet Hyg Part 4

Sellers, R F 1955 Growth and titration of the viruses of foot and mouth disease and vesicular stomatitis in kidney monolayer tissue culture Nature London 176 4481 547

Shahan, M S 1954 Present situation on foot and mouth disease Military Surgeon 114 444

Skinner, H H 1951 Propagation of strains of foot and mouth disease virus in unweaned white mice Proc Roy Soc. Med 44 104

——— 1953 *One week old mice as test animals in foot and mouth disease research* Proc 15th Internat Vet. Cong 1 195

——— 1954 Infection of chickens and chick embryos with the viruses of foot and mouth disease and vesicular stomatitis Nature London 174 1052

Stockman, S and Minett, F E 1926 Experiments on foot and mouth disease Jour Comp Path and Therap 39 231

Terbruggen, F 1932 Über die Haltbarkeit des Maul und Klauenseuchevirus in Milch und Molkereiprodukten Deutsch tierarzt Wschr 40 129 529

Thomas J A Thiéry, J P Salomon, L and Salomon L 1952 La culture du virus de la fièvre aphteuse en trés grande quantité sur embryone spécifique géant chez la vache C R Acad Sci Paris 235 520

Traub E and Mohlmann, H 1943 Typesbestimmung bei Maul und Klauenseuche mit Komplementbindungsprobe Zentralbl f Bakt I Abt Orig 150 289

——— and Schneider B 1948 Züchtung des Virus der Maul und Klauenseuche im bebrüteten Huhnerei Zeitschr f Naturf (B) 3 178

Traum, J 1934 Foot and mouth disease Specific treatment eradication and differential diagnosis 12th Internat Vet Cong Proc p 87

——— 1951 Foot and Mouth Disease *In* Cecil and Loeb Textbook of Medicine 8th ed W B Saunders Co Philadelphia p 49

Trautwein, K 1925 Zur Frage der Einschlusskorperchen bei Maul und Klauenseuche Arch wiss parkt Tierheilk 52 475

——— 1929 Maul und Klauenseuche Ergeb Hyg Bakt Immunitatsforsch u Exper Therap 10 561

——— and Reppin, K 1928 Versuche zur Disinfektion bei Maul und Klauenseuche mit schwefliger Saure sowie mit Natronlauge Arch f Tierheilk 58 96

U.S.D.A 1926 Foot and Mouth Disease Circ. No 400

———. 1943 Instructions for employees engaged in eradicating foot and mouth disease

U.S.D.A Release 1947 Summary developments in the Mexican outbreak of foot and mouth disease Jan 28 1947

——— 1953 No 1279-53 May 28 1953

——— 1954a No 999-54 April 14 1954

——— 1954b No 3219-54 December 22 1951

Vallée H and Carré, H 1922 Sur la pluralité du virus aphteux C R Acad Sci Paris 174 (II) 1498

Vetterlein W 1954 Das klinische bild der Maul und Klauenseuche beim Menschen auf gestellt aus den bisher experimentell gesicherten Erkrankungen Arch f Exper Veter märmed 8 (5) 541-4

Wagener, K 1928 Jauche und Jauchebeseitigung und ihre hygienische Bedeutung fur die Bekämpfung der Maul und Klauenseuche Arch wiss prakt Tierheilk 58 217

Waldmann, Ö 1930 Über Maul und Klauenseuche Proc. 11th Internat Vet Cong 2 37

——— and Köbe, K 1938 Die aktive Immunisierung des Rindes gegen Maul und Klauenseuche. 3rd Internat Cong Microbiol Rep Proc., 1939-40 p 360

——— and Nagel, H C 1939 Die Maul und Klauenseuche Handbuch der Viruskrankheiten Gustav Fischer Jena 1 385

——— and Pape J 1920 Die künstliche Übertragung der Maul und Klauenseuche auf das Meerschweinchen Berl tierarztl Wschr 36 519

——— and ——— 1921 Experimentelle Untersuchungen über Maul und Klauenseuche Berl tierarztl Wschr 37 349 449

——— and Trautwein, K 1926 Experimentelle Untersuchungen über die Pluralität des Maul und Klauenseuchevirus. Berl tierärzt Wschr 42 569

——— and Pyl, G 1951 Die Persistenz des Maul und Klauenseuchevirus im Korper durchseuchter Tiere und seine Ausscheidung Zentralbl f Bakt I Abt Orig 121 19

the brains of animals from both the Mc
Nutt and the Ray outbreaks

Accounts of the clinical signs exhibited
by baby pigs suffering from pseudorabies
are numerous in the foreign literature and
excellent descriptions have been given by
Hirt (1935), Lazlo (1938), Lamont and
Shanks (1939), Carneiro and Cardim
(1947), and Gordon and Luke (1955)
One of the most complete of these is the
account given by Gordon and Luke
(1955) of the disease on a farm in North
ern Ireland and I shall quote from their
published description of the clinical pic
ture of pseudorabies as they saw it among
young pigs on this farm

The disease affected piglets from a few days
old up to four weeks In general the young
er the pigs at the onset of symptoms the more
widespread was the resultant clinical disease in
the litter In all cases the onset was usually
sudden and in many instances the whole litter
became affected and died within 48 hours
Among some of the older litters one or two pig
lets remained apparently healthy and survived
while the remainder of the litter died

In the older pigs there were clear cut symp
toms of involvement of the nervous system,
there was a degree of incoordination affecting
usually the hind quarters and piglets thus af
fected tended to move sideways This inco
ordination progressed rapidly and complete
paralysis soon supervened though in a few
cases periods of remission in the severity of the
symptoms were seen Spasmodic twitching of
groups of muscles paddling movements and
fine muscular tremors were all exhibited in
varying severity by affected piglets Convulsive
fits were seen but not commonly and a tend
ency to carry the head to one side developed
in some animals In the younger pigs the symp
tomatology was more confused and distinct
nervous symptoms were not always apparent
There was a tendency for such young pigs to
stand with their backs arched and the ears
flattened along the sides of the neck The coat
lost its sleek appearance and the hair tended
to stand on end

Many of the affected pigs developed snoring
respirations with marked abdominal type of
breathing This respiratory distress was not
associated with any evidence of pneumonia
post mortem and probably indicated involve
ment in the disease process of the respiratory
centre in the brain Some of the affected pigs
showed signs of slight ocular discharge Some
surprising recoveries were recorded even in pigs

completely paralysed In the final stages af
fected pigs tended to creep into corners away
from the rest of the litter The temperature of
affected pigs varied greatly, up to 106°F being
recorded in a few cases but in most sick pigs
it was within normal limits or raised a degree
or two Many badly affected pigs had sub
normal temperatures There was evidence, how
ever that a temperature reaction preceded the
onset of clinical disease The weaned pigs in
the same house with the recently farrowed sows
and their litters at no time showed any evi
dence of a sickness even though heavy losses
were taking place in the young piglets

Pseudorabies as it occurs naturally in
older swine in the United States is a 'si
lent subclinical infection Affected ani
mals are not noticed by their owners to
be ill It is of veterinary importance
largely from the standpoint of the role
that infected swine play in the transmis
sion of pseudorabies virus to cattle The
disease also appears to be extremely mild
in older pigs in Brazil (Carneiro 1941)
and in Northern Ireland (Lamont, 1946)

Under experimental conditions in the
laboratory, swine 7 weeks of age or older
can be infected with pseudorabies virus
when inoculated subcutaneously, intra
muscularly, intranasally, intracerebrally, or
when fed (Shope, 1935a) Fatal infections
have, however, been regularly produced
only in intracerebrally inoculated swine,
death occurring in from 36 to 96 hours
with signs indicative of an encephalitis
Administration of virus by all other routes
has resulted in a more or less mild disease
and, among 44 swine infected by ways
other than intracerebral, no fatal cases
have resulted and none have been seriously
ill

The salient clinical feature of swine
pseudorabies induced by subcutaneous in
oculation is a febrile reaction of variable
duration (1 to 8 days) during which the
animals exhibit a transient depression and
inappetence Infection by routes other
than subcutaneous results in a similar
clinical picture with but minor variations
Perhaps the most significant variation
from a practical standpoint, is to be seen
in animals to which virus is administered
intramuscularly in the ham or deep in the

Chamberland candles when adequate care is taken to clarify the infectious suspension of gross *debris* before filtration It can be inactivated by heat, formaldehyde, or ul traviolet light, though it is relatively re sistant to the action of phenol Braga and Faria (1934) found that the virus still showed some infectivity after 40 days' ex posure to 3 per cent phenol, and Koves and Hirt (1934) stated that filtrates of the virus were still virulent after 32 days' exposure to 0 5 per cent phenol This rel ative resistance of pseudorabies virus to phenol is important in hog cholera im munization since the pseudorabies virus may occasionally be present as a contami nant of commercial hog cholera virus and not be inactivated by the phenol present in this product When this happens, swine that are vaccinated with the contaminated hog cholera preparation develop pseudo rabies, a disease of some consequence in young pigs, as will be pointed out later in this chapter *Cases of suspected accidental infection of swine with pseudorabies through the medium of contaminated hog cholera biological products have been re ported* by Burggraaf and Lourens (1932) in Holland, Koves and Hirt (1934) in Hungary, and by McNutt and Alice (1942) and McNutt and Packer (1943) in the *United States*

Pseudorabies virus was first cultivated by Traub (1933) in rabbit testicle, guinea pig testicle, and chick embryo media La ter Glover (1939), Burnet *et al* (1939), and Bang (1942) grew it on the chorio allantoic membrane of the developing egg Beladi and Szollosy (1955) observed that in monolayer chick-embryo cell tissue cul tures, pseudorabies virus produced circum scribed plaques in proportion to the titer of virus infecting the cultures and that plaque counts could, therefore, be used in determining the number of infective parti cles in a virus suspension

Pseudorabies virus has been observed by many investigators as a cause of clinical illness under *natural conditions* in cattle cats, dogs, horses, rats and swine Ex perimentally it is fatally pathogenic for rabbits, guinea pigs, mice and, when given cerebrally, chickens and ducks

CLINICAL SIGNS

Pseudorabies occurs as a clinically ap parent disease among swine in the United States only in young animals Older ani mals, though highly susceptible to the virus, undergo an inapparent 'silent in fection and are ordinarily not observed to be ill To judge from reports in the liter ature porcine pseudorabies is of consider ably *more veterinary importance* in the Old World than it is in either the United States or in South American countries In Northern Ireland and in parts of Eu rope, especially in Russia and in the Balkan countries, pseudorabies in young pigs is a highly fatal and quite widely prevalent infection and the disease, even in older animals, is associated with clinical illness and some mortality

So far as I am aware, only two naturally occurring outbreaks of porcine pseudori bies have been observed in the United States (Ray, 1913 McNutt and Packer 1943) In both of these incidents, only young pigs were involved In one of the outbreaks (McNutt and Packer, 1943), 105 out of 176 baby pigs succumbed of the disease Dr McNutt has told me (personal communication) that newborn pigs die very rapidly of pseudorabies, passing from apparent normalcy to coma in less than an hour, and dying a few hours after they become comatose In very young pigs, distinct signs of central nervous system in volvement are not seen

The other outbreak, described by Ray (1913) had killed 84 out of 190 pigs, that were 1 to 2 weeks of age, at the time Dr Ray s observations were made Three sick animals from the group were carefully ob served One of these, less severely affected showed incoordination and excitability This animal improved gradually, and ap parently recovered completely in a few days The other two baby pigs exhibited a progressive paralysis, were excitable and finally became prostrate and died Pseudo rabies virus was demonstrably present in

TABLE 131

TRANSMISSION OF PSEUDORABIES IN SWINE BY CONTACT*

Day of Experiment	Experiment 1		Experiment 2		
	Swine 1469 Infected With Virus Intramuscularly	Swine 1466 Infected by Exposure	Swine 1483 Infected With Virus Intramuscularly	Swine 1482 Infected by Exposure	Swine 1477 Infected by Exposure
1					
2	X	{Placed in pen with (Swine 1469)}	X	{Placed in pen with (Swine 1483)}	
3	X	←	X	←	
4	X	0	X + (135 hrs)	0	
5	X + (104 hrs)	0	X	0	
6	+ (117 hrs)	0		0	
7		0			
8	–	0	+ (119 hrs)	X + (78 hrs)	
9	–	0		X + (71 hrs)	
10	–	0		X + (63 hrs)	
11	–	+ + (166 hrs)		X + (71 hrs)	
12		X + (98 hrs)		X + (70 hrs)	{Placed in pen with (Swine 1482)}
13		+ (79 hrs)		X + (72 hrs)	← 0
14				X + (89 hrs)	+ (126 hrs)
15		+ (82 hrs)		+ (95 hrs)	X + (101 hrs)
16				+ (87 hrs)	X + (85 hrs)
17				+ (113 hrs)	X + (71 hrs)
18				+ (122 hrs)	X + (63 hrs)
19				0	
20				–	
21				–	
22					

* key to symbols
X = temperature elevation to fever level (104° F or higher)
0 = nasal passages not lavaged
– = virus absent in nasal washings—test rabbit survived
+ = virus present in nasal washings—test rabbit died of pseudorabies death

(Hours in parentheses indicate time elapsing between inoculation and death.)

axillary space   On the 3rd to the 5th day following infection, such swine develop a transient or permanent flaccid paralysis of the inoculated leg   Aside from the paralysis, the disease seen in these swine is similar to that exhibited by subcutaneously infected animals

In connection with the paralysis caused by injection of pseudorabies virus into the ham or axilla, McNutt and Alice (1942) and McNutt and Packer (1943) observed paralysis in large numbers of swine to which a serial of commercial hog cholera virus contaminated with pseudorabies virus, had been given in the process of immunization against hog cholera

Pseudorabies, relatively, is a highly contagious disease in swine, both in the field and under laboratory conditions   The incubation period on pen exposure varies from 3 to 11 days, depending upon the stage of the disease in the infected animal when the normal animal is placed in the pen   The contact disease is mild and indefinite like that induced by subcutaneous inoculation, and animals infected by exposure are never seriously ill   Virus spreads from pig to pig, probably by way of the nose   Virus is demonstrably present in the noses of contact infected swine for variable lengths of time, from as short a period as 2 days to as long as 11 days   Typical contact experiments are depicted in Table 13 1

The field disease as it occurs under natural conditions in the United States in pigs of weanling age or older is apparently like that just described for swine infected by contact under experimental conditions — mild and ordinarily not recognized clinically   It remains unrecognized in its swine host and its presence in a community becomes known only when the virus spreads from its "silent" porcine host to cattle in which a uniformly fatal pseudorabies results (Shope, 1935a, Carneiro, 1939)

The determination of the incidence of pseudorabies virus neutralizing antibodies

in sera collected from swine in the mid western United States indicates that porcine pseudorabies is prevalent in that region to a startling extent (Shope, 1935b), even though seldom recognized clinically A similar situation has been described by Carneiro (1941) and Carneiro and Cardim (1947) as prevailing in parts of Brazil where serological surveys of swine sera have revealed a high incidence of pseudorabies virus neutralizing antibodies, indicating a widespread prevalence of unrecognized porcine pseudorabies in that country also

Table 13 2 gives the results of pseudorabies virus neutralizing antibody determinations on 23 different serials of commercial anti hog cholera serum randomly selected from that being used by veterinary practitioners in eastern Iowa   The high incidence of antibodies found is indicative of the widespread prevalence of unrecognized porcine pseudorabies in the Mid west

Though pseudorabies does not appear to be a clinically recognized disease in adult swine in the United States, the same statement cannot be made about its many festations in certain European countries Burggraaf and Lourens (1932) described an outbreak of pseudorabies in Holland in a drove of 600 to 700 swine, in which the disease, while benign and ill defined in most animals, was nevertheless characterized by epistaxis, vomition, and nervous symptoms in the more severely affected hogs   Nearly all eventually recovered Those that died apparently succumbed in convulsions, some showing a hemorrhagic enteritis at death   Koves and Hirt (1934) have indicated that pseudorabies in Hungary is a major problem among swine   The disease they have described as resulting from infection with the pseudorabies virus appears to resemble somewhat our swine influenza   The illness is usually ushered in by a fever of up to 106° F, the animals become weak, their lust for food is diminished, and many of them vomit.   Most cases recover in a day or two, but some

progress and present evidence of lung involvement Diarrhea is stated to be common A small percentage of the infected swine develop signs referable to the central nervous system They become listless tremble, some have convulsions, and paralysis may supervene Some of the animals salivate The swine that eventually succumb usually show marked signs of central nervous system involvement Itching and biting at localized areas has not been observed in swine in Hungarian outbreaks Uzlova (1955) has described outbreaks of pseudorabies in adult swine in Russia, in which the symptoms were very similar to those outlined for Hungarian outbreaks of the disease

There is no apparent reason to explain why a more severe type of swine pseudorabies is seen in European countries than in the United States Variations in husbandry and feeding practices perhaps may account for some of the difference Also it may be possible that we are not recognizing the identity of all of our outbreaks of porcine pseudorabies, or are confusing them with other conditions

PATHOLOGICAL CHANGES

Porcine pseudorabies presents almost nothing to see grossly at necropsy Koves and Hirt (1934) described secondary lesions in the throat resulting from paralysis of the pharynx and observed pulmonary edema to be common in fatal adult cases They also described lesions in the gastrointestinal tract, largely inflammatory, but sometimes of such extent as to result in massive erosion of the intestinal mucous membrane with croupous exudates overlying the eroded areas None of the investigators who have observed fatal cases of pseudorabies in baby pigs have described the gross pathology, thus it may be assumed that in such animals there is no grossly evident pathology to see at necropsy

HISTOPATHOLOGY

The histopathology of porcine pseudorabies has been described by Hurst (1933)

and Hirt (1936) Hurst, studying swine that had been inoculated intracerebrally with virus, noted meningitis, perivascular infiltration often associated with diffuse proliferation of microglial cells in the surrounding tissues and microglial proliferation and cellular infiltration in the superficial cortical zone immediately beneath the pia arachnoid as salient findings Nerve cell changes were slight and only in the densest tissue foci did some neurons manifect severe degenerative phenomena culminating in death and neuronophagia Hurst could find no nuclear inclusions in porcine pseudorabies despite the fact that such inclusions are present in the cells of the nervous tissue in all other species of animals with pseudorabies Hirt studied material from baby pigs as well as adult swine and remarked that a non purulent meningo encephalitis with perivascular filtration was common Like Hurst he could demonstrate no cell inclusions From Hirt s description, it may be gathered that nerve cell damage is somewhat greater in young pigs than was found by Hurst in older animals, since Hirt refers to a pronounced neuronophagia and marked glial proliferation in the ventral horns of the gray matter of the cord

DIAGNOSIS

A disease showing clinical signs as rarely as does porcine pseudorabies should be well down on the list of suspected conditions that one would have to consider in observing a drove of sick swine However, it should be suspected in any instance in which newborn or young swine sicken rapidly, and die either in coma or with central nervous system signs The diagnosis can be established and verified easily and rapidly by the injection of rabbits subcutaneously with brain aseptically obtained from the dead baby pig The brain should be prepared in approximately 10 per cent suspension in sterile physiological saline and injected in 1 or 2 ml amounts subcutaneously into the test rabbits If pseudorabies virus is present, the inocu

TABLE 13 2

NEUTRALIZATION OF PSEUDORABIES VIRUS BY COMMERCIAL ANTI-HOG CHOLERA SERA

Anti-Hog Cholera Serum Sample	Number of Swine Represented in Sample	Results With Different Amounts of Serum Mixed With 100 mg of Pseudorabies Virus and Administered Subcutaneously to Guinea Pigs	
		1 0 cc	0 1 cc
1	50	No illness *	Died—58 hrs
2	146	No illness	No illness
3	152	No illness	Died—95 hrs
4	151	No illness	No illness
5	150	No illness	No illness
6	148	No illness	Died—73 hrs
7	48	No illness	Died—104 hrs
8	158	No illness	No illness
9	110	Died—95 hrs	
10	170	No illness	No illness
11	103	No illness	No illness
12	100	No illness	No illness
13	129	No illness	No illness
14	169	No illness	No illness
15	106	No illness	Died—70 hrs
16	165	No illness	Died—91 hrs
17	?	Died—69 hrs	
18	?	No illness	
19	85	No illness	No illness
20	152	No illness	Died—84 hrs
21	103	No illness	Died—68 hrs
22	126	No illness	No illness
23	?	No illness	No illness

Control Sera †

Swine 1237 ‡		Died—68 hrs	Died—63 hrs
Swine 1237		Died—66 hrs	Died—61 hrs
Swine 1237		Died—52 hrs	Died—70 hrs
Swine 1444		Died—64 hrs	
Swine 1444		Died—63 hrs	
Swine 1444		Died—73 hrs	
Swine 1449		Died—58 hrs	
Swine 1449		Died—70 hrs	
Swine 1229		Died—95 hrs	
Swine 1229		Died—70 hrs	
Swine 1446		Died—58 hrs	
Swine 1446		Died—70 hrs	

* Failure of 1 0 cc. of serum to neutralize the pseudorabies virus indicates that the group of swine furnishing the serum either had been entirely free of pseudorabies or that the incidence in the group had been less than 5 per cent (samples 9 and 17)

Neutralization of the virus by 1 0 cc but not by 0 1 cc of serum is interpreted as indicating a previous pseudorabies infection involving at least 5 per cent of the swine supplying the serum (samples 1, 3, 6, 7, 15, 16, 18, 20, and 21)

Neutralization of the virus by both 1 0 and 0 1 cc amounts of serum is interpreted as indicating a previous pseudorabies infection of upwards of 50 per cent in the group of swine furnishing the serum (remaining 12 samples)

† 0 5 per cent phenol was added to all control sera to make them comparable to the commercial anti hog cholera sera

‡ All swine furnishing control sera were from Institute stock

reservoir from which associated cattle are infected Since the disease in cattle, in contrast to that in swine, is uniformly fatal, the presence of a pseudorabies virus infection in swine that are following cattle can be disastrous for the associated cattle Because of this definite association, cattle should be removed from contact with any swine drove in which pseudorabies is suspected Furthermore, in the event that pseudorabies occurs in cattle that are running with swine, the very first precaution ary step to be taken in order to limit the spread of the disease among the cattle is to remove them from all contact with those swine

RABIES IN SWINE

True rabies in swine, to judge from the paucity of literature on the subject, appears to be a rather rare condition Swine are susceptible, however, to infection with the rabies virus When infected, they show a clinical picture similar to that of rabies in other animals Intense excitement, followed by weakness and, eventually, paralysis, usually precedes death in porcine rabies The incidence of rabies in swine in the United States is much lower than in dogs, for instance, for the years 1952 and 1953, when there were approximately 11,000 cases of rabies in dogs, there were only 70 cases in swine

REFERENCES

AUJESZKY, A 1902 Über eine neue Infektionskrankheit bei Haustieren Zentralbl f Bakt Parasit u Infekt 32 353
BANG, F B 1942 Experimental infection of the chick embryo with the virus of pseudorabies Jour Exper Med 76 263
BÉLÁDI I O AND DVANOVICS G 1954 Labóratoriumi állatok immunizálása ultraibolya fénnyel inaktivált Aujeszky féle virusal Mag allator Lapja 9 255
—— AND SZÖLLÖSY, E 1955 Production of plaques in monolayer tissue culture by Aujeszky disease (pseudorabies) virus Acta Microbiol Acad Sci Hung 3 213
BRAGA, A, AND FARIA, A 1934 Paralysia bulbar infectuosa Bol Instit Vital Brazil No 16 (Fev ereiro) 1
BURGGRAAF, A AND LOURENS, L F D E 1932 Infectieuse Bulbair Paralyse (Ziekte van Aujeszky) Tijdschr v Diergeneesk 59 981
BURNET, F M LUSH, D AND JACKSON, A V 1939 The propagation of herpes B and pseudorabies viruses on the chorioallantois Australian Jour Exper Biol and Med Sci 17 35
CARNEIRO V 1939 Anticorpos neutralisantes do virus da doença de Aujeszky em soros de porcos no Brasil Arq Inst Biol São Paulo 10 305
—— 1941 Adoença de Aujeszky em suinos no Brasil nos focos de epizootias em bovinos — pela pesquisa de anticorpos neutralisantes do virus Arq Inst Biol Sao Paulo 12 213
—— AND CARDIM, W H 1947 A doença de Aujeszky em suinos no Brasil Arq Inst. Biol Sao Paulo 18 243
GLOVER, R E 1939 Cultivation of the virus of Aujeszky s disease on the chorio allantoic membrane of the developing egg Brit. Jour Exper Path 20 150
GORDON, W A M, AND LUKE D 1955 An outbreak of Aujeszky s disease in swine with heavy mortality in piglets illness in sows and deaths in utero Vet Rec 67 591
HANSON R P 1954 The history of pseudorabies in the United States Jour Amer Vet Med Assn 124 259
HIRT G 1935 Beitrage zur Aujeszkyschen krankheit der Saugferkel Arch wiss prakt Tierheilk 70 86
—— 1936 Histologische Veranderungen des Zentralnervensystems bei der Aujeszkyschen Krankheit Arch wiss prakt Tierheilk 70 323
HURST E W 1933 Studies on pseudorabies (Infectious bulbar paralysis mad itch) I Histology of the disease with a note on the symptomatology Jour Exper Med 58 415
KOVES J AND HIRT, G 1934 Über die Aujeszkysche krankheit der Schweine Arch wiss prakt Tierheilk 68 1
LAMONT H G 1946 Observations on Aujeszky s disease in Northern Ireland Vet Rec. 58 621
—— AND SHANKS P L 1939 An outbreak of Aujeszky s disease amongst pigs Vet Rec 51 1407
LAZLO H 1938 Boala lui Aujeszky ca epizootie purcellor sugari Rev Med Vet Bucharest 50 301
McNUTT, S H AND ALICE F J 1942 Doença de Aujeszky (pseudoraiva) em suinos Bol d Soc Bras Med Vet 11 61

lated rabbits will begin biting and scratching at the site of inoculation within 48 to 72 hours, and will ordinarily be dead within 24 to 36 hours of the time they began itching. Necropsy of such test animals will reveal a raw, bloody, denuded area of skin at the site of injection. Pseudorabies virus will be present in the brains of such rabbits and can be passed serially in this host using brain as the inoculum.

IMMUNITY

Swine acquire a solid immunity following infection with pseudorabies virus, and specific virus neutralizing antibodies appear in their blood serum upon recovery. As mentioned earlier, most serials of hog cholera hyperimmune serum produced in the midwestern United States contain neutralizing antibodies for the pseudorabies virus due to the fact that donors of such serum have earlier undergone an attack of pseudorabies.

Pseudorabies in swine is of such minor importance in this country that no effort has been made to develop an immunizing product. However, in Russia and the Balkans, where the disease is apparently of greater importance, several vaccines have been prepared and used. Among these, Beládi and Ivánovics (1954) spoke of the use of a pseudorabies vaccine in which the virus had been inactivated by ultraviolet light. Popovici et al (1955) have used a vaccine comprised of virus from the brains of infected sheep adsorbed on aluminum hydroxide.

TREATMENT

There is no known specific treatment for pseudorabies. Since the disease is contagious in swine, isolation of sick and known infected animals should be practiced where possible.

EPIZOOTIOLOGY AND CONTROL

A disease as highly contagious for swine as pseudorabies may be expected to spread through a drove by contact of sick animals with normal animals. How the virus tides over between outbreaks and how it is spread from farm to farm are not known. A number of investigators have hypothesized that infected rats may be responsible for carrying the disease from one farm to another, but evidence for this is not very convincing.

It seems most likely that the extremely low incidence of pseudorabies in baby pigs in the United States may be the direct result of the very high incidence of infection of swine herds here. Most adult swine on middle western farms, to judge from the high incidence of neutralizing antibodies found in the serum of such animals, are immune by virtue of previous infection with the virus. The offspring of immune sows would be expected to acquire sufficient passive protection from their mothers to protect them during the period when they are most highly susceptible to fatal infection. The baby pigs that do come down with pseudorabies and suffer fatal infections must be those from mothers who themselves are still susceptible to infection. Hence, clinically apparent outbreaks of pseudorabies in newborn pigs might be visualized as the result of the introduction of the virus into a fully susceptible drove of swine at about the time that farrowing was taking place. Under such circumstances, the sows and the older pigs on the place would be expected to undergo a mild subclinical pseudorabies infection and probably serve as the source of infection for the newborn baby pigs, which, because of their age, would undergo a clinically more severe and fatal virus infection. The extremely low incidence of pseudorabies in baby pigs in the United States suggests strongly that such epidemic situations seldom occur here.

As mentioned earlier, one of the most important features of porcine pseudorabies as it occurs in the United States, concerns the role that swine play in the epidemiology of the disease in cattle. It seems most likely that swine serve as the virus

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CHAPTER 14

Porcine Encephalomyelitis
(Teschen Disease)

This specific viral infection of the central nervous system of swine was first recognized in the region of Teschen, Czechoslovakia, from which comes its common name Other synonyms are encephalomyelitis enzootica suum, poliomyelitis of swine, Bohemian pest, meningo encephalomyelitis suum *Teschener Krankheit, an steckende Schweinelahmung,* and *meningo encephalomyelite enzootique du porc* Treffny described and named the disease in 1929, but it is possible that Klobuk may have observed cases in Moravia as early as 1913 (Kaplan and Meranze, 1948) The disease is still enzootic in Czechoslovakia and has been reported in central and western Europe on numerous occasions Severe outbreaks have appeared in Madagascar (Pilet, 1952) The disease is not known to occur in the Western Hemisphere

ETIOLOGY

The virus of Teschen disease is found during the course of the disease in the brain and spinal cord, it may appear in the feces and, transiently, in the blood It is rarely found elsewhere The virus is quite resistant to drying but is destroyed by heating at 60° C for 20 minutes or at 70° C in 30 minutes The agent being one of the smaller viruses is filtrable

through Berkefeld N, V, and W filters Ultrafiltration experiments (Patocka *et al* 1952) indicate the size of the virus particle to be about 25 mμ The virus is active at pH 2 5 to 13, but increased virulence is reported at pH 8 to 11 The virus is not affected by suspension in ether It does not infect species other than swine as far as is known, neither does it grow in developing chick embryos, but it has been reported to multiply and produce cytopathogenic effects in cultures of embryonic swine tissues (Larski 1955, Mayr and Schwobel, 1956) The virus readily produces infection after intracerebral inoculation, less reliably following intranasal or intraperitoneal injection, but does not result in disease after intravenous injection

The virus of Teschen disease does not have the property of hemagglutination (Nam *et al*, 1955b)

CLINICAL FEATURES

The incubation period following experimental exposure to the virus by intracerebral or intranasal routes averages about 6 days but may range from 1 to 28 days, depending upon the amount of virus given The incubation period after natural exposure is not well documented but presumably falls within the time limits observed in experimental infection

In swine populations which have not been previously exposed to the virus, the

McNutt, S H , and Packer A   1943 Isolation of western equine encephalomyelitis and hog cholera viruses from supposedly hog cholera immune swine Vet Med 38 22

Popovici, I , Taga, M and Berbinschi, C   1955 Vaccinarea contra bolii lui Aujeszky la porci Anu Inst Pat Igien Anim , Bucharest 5 123

Ray, J D   1943 Pseudorabies (Aujeszkys disease) in suckling pigs in the United States Vet Med 38 178

Sangiorgi, G   1914 La filtrabilita del virus della pseudorabbia Pathologica 6 201

Schmiedhoffer J   1910 Pathologie der infektiosen Bulbar Paralyse (Aujeszykschen Krank heit) Zeitschr f Infektionskrankh , parasit Krankh u Hyg d Haustiere 8 383

Shope, R E   1931 An experimental study of mad itch with especial reference to its relation ship to pseudorabies. Jour Exper Med 54 233

——   1935a Experiments on the epidemiology of pseudorabies I Mode of transmission of the disease in swine and their possible role in its spread to cattle Jour Exper Med 62 85

——   1935b Experiments on the epidemiology of pseudorabies II Prevalence of the disease among middle western swine and the possible role of rats in herd to herd infections Jour Exper Med 62 101

Traub E   1933 Cultivation of pseudorabies virus Jour Exper Med 58 663

Uzlova L M   1955 Nervnaia forma bolezni Aueski u vzroslykh svinei Veterinariya Moscow 32 83

tention of the pathologist are the neuronophagic nodules evident in the gray matter. These nodules are made up of dense or loose aggregations of cells which can be detected under low magnification (Figs. 14.1, 14.2). The cells which make up these nodules have round to ovoid nuclei which are either finely or densely stippled with chromatin. The cytoplasm is indistinct in most of them when stained by Nissl's method, but a hematoxylin and eosin stain sometimes brings out a cell membrane which makes each cell appear discrete. In many cells the cytoplasm still cannot be distinguished, even in hematoxylin and eosin preparations.

The presence of partially phagocytized fragments of neurons in the center of some of these collections of cells clearly establishes them as neuronophagic nodules. In many such nodules, however, the relationship to necrotic neurons is not clearly evident, prompting some writers to refer to them simply as "cell nodules."

The exact identity and origin of the cells that make up these nodules is currently a subject of dispute among neuropathologists. One traditional view is that

these cells are derived from glial cells; therefore, the aggregations are referred to as "glial nodules." Another opinion holds that the cells are derived from glial (especially microglia) and adventitial cells of the blood vessels; hence, the nodules are considered "glial-mesenchymal" in origin. A third theory contends that all of the cells result from activation of vascular adventitia; the nodules are therefore considered to be solely mesenchymal in origin. The fourth prominent contention is that these cells all result from infiltration, proliferation, and modification of lymphocytes. Present evidence appears inadequate to establish clearly any one of these theories concerning the nature of these cells, but this uncertainty does not preclude the recognition of the nodules and their utilization in the diagnosis of Teschen disease. In this chapter, the terms *neuronophagic nodule* and *cell nodule* will be used, the latter when degenerating neurons do not appear to be the nidus for the nodule.

Neurons. The changes which occur in the nerve cells are obviously very impor-
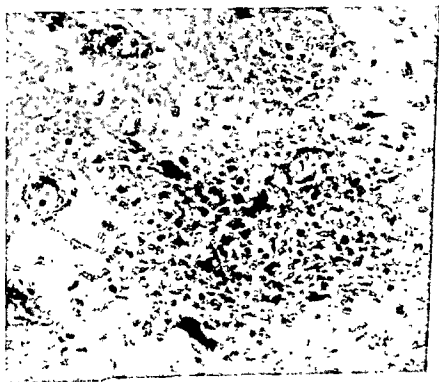
FIG. 14.1—Neuronophagic nodule in a thalamic nucleus, brain of a pig with experimental Teschen disease. A.F.I.P. 514663. Nissl's stain. X 400.

disease may appear in one individual, then spread through the rest of the herd until almost all are sick In other situations, the disease may appear in successive waves, weeks or months apart, but eventually involves most animals in the herd In enzootic areas, the disease reportedly may assume a sporadic character, affecting only individual animals on a farm

The initial signs are usually fever (104° F to 106° F or higher) with slight incoordination of the rear limbs, lassitude, and anorexia A stage of irritability usually follows in a few hours or days, then stiffness in the extremities appears and the animal may fall repeatedly Some animals may exhibit a stiff, tripping gait and muscular rigidity, with the forelegs being placed forward and the hind legs drawn backwards In severe cases tremors, nystagmus, violent clonic convulsions, prostration and coma appear and persist for many hours Convulsions, accompanied by loud squealing, may be set off by a sudden loud noise In some cases the most severe and enduring signs are stiffness and opisthotonus Smacking the lips and grinding the teeth are observed in some animals Others may chew on objects and occasionally squeal as if in pain

As paralysis appears and becomes the dominant feature, the animal may sit upon its haunches like a dog or fall to its side, where it remains helpless Stimulation by loud sounds or simply touching the animal may cause severe opisthotonus, accompanied by thrashing movements of the forelegs This struggle may cause the recumbent animal to propel himself around in a circle The patellar reflex is diminished or lost at this stage and cutaneous sensitivity is usually lowered or lost in some part of the body The voice may be toneless or entirely lost Constipation is usual but the appetite often remains good

Vesicular eruptions have been reported to appear on the snout but these have not been shown to be specifically related to Teschen disease

The course of the disease is most often

acute, death occurring within 3 or 4 days after onset Only a few cases are peracute the animals dying within 24 hours Some animals have been known to survive many months with careful nursing, but residual paralysis and atrophy of muscles are evident Mildly affected animals may recover completely Inapparent infections also are known to occur

The disease is most likely to affect young swine in enzootic areas, but swine of all ages and breeds are fully susceptible Animals which have survived a mild or inapparent infection usually have a degree of immunity to subsequent infections This may explain the lowered susceptibility of older swine in enzootic regions

PATHOLOGIC CHANGES

Gross Lesions

No specific gross lesions are recognizable in animals dead of porcine encephalomyelitis Atrophy of muscles may be observed, however, in paralyzed animals in which the disease has undergone a prolonged course

Microscopic Lesions

The principal effect of the virus in the central nervous system is upon neurons therefore, the recognizable lesions are found for the most part in the gray matter The neurons undergo degenerative changes leading to necrosis which in turn are followed by aggregation of cells near the sites of injury In nervous tissue, because the range of reaction to injury is very limited, differentiation of lesions must depend not only upon the precise details of the tissue response but also upon the anatomic distribution of the lesions In Teschen disease the details of the cellular changes in the lesions and their anatomic location are important to the differential diagnosis of the disease and hence will be described in detail These changes have been carefully studied, particularly by Manuelidis _et al_ (1951)

Neuronophagic nodule The microscopic lesions which first attract the at-

FIG. 14.3—Perivascular accumulation of lymphocytes in Virchow-Robin spaces. Occipital cortex. A.F.I.P. 514663. Nissl's stain. X 35.
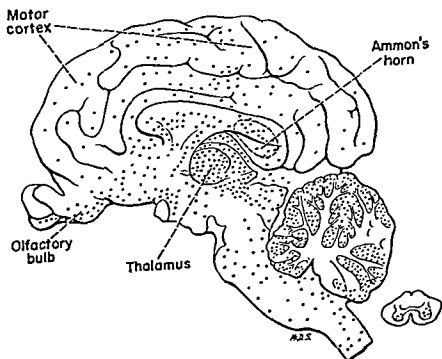
FIG. 14.4—Distribution of lesions of Teschen disease. Each dot represents a lesion. After Manuelidis et al. (1954).

tant in this disease. Degeneration and death of neurons is at least one factor underlying the formation of neuronophagic nodules and is the basis for the symptoms. The recognition of lesions in the neurons and their differentiation from artefact are often difficult, especially in early stages, and require both good technical preparations and careful study. Affected neurons appear shrunken and distorted, their nuclei may be absent, and Nissl granules may be densely stained or absent from the cytoplasm. Small nuclei of satellite cells usually gather adjacent to the affected neuron, and large numbers of these small cells may assemble to make up a frank neuronophagic nodule as described above.

Perivascular changes. Accumulation of cells around the smaller vessels may be a prominent feature in porcine encephalomyelitis although it is neither specific nor limited to this disease. The pia mater is anatomically deflected around each blood vessel to form a barrier between the wall of the vessel and the brain parenchyma. The interval between the vessel wall and this layer of pia, called the Virchow-Robin space, remains a potential cavity until it is filled with gas, fluid, or cells. Leuko-

cytes, stimulated to penetrate the arterial or venous wall, often accumulate in the Virchow Robin space to form a "collar" or "cuff" of cells around the blood vessel. This perivascular cuffing is one of the least specific reactions in nervous tissue but may, however, be a prominent feature in the microscopic picture. Lymphocytes are the most numerous cells in this perivascular exudate (Fig. 14.3), but plasma cells and neutrophils may also be seen. Blood vessels within gray matter may be affected, but the most prominent changes are found around vessels in white matter adjacent to the grey masses. Perivascular cuffing is a feature of all of the viral encephalitides but also may be found in the brain adjacent to areas of ischemic necrosis, old hemorrhage, or almost any injury to brain parenchyma.

Distribution of lesions. The lesions of Teschen disease are concentrated in the gray matter of the ventral spinal columns, cerebellar nuclei and cortex, the brain stem, and, to a lesser degree, motor cortex (Fig. 14.4), but, unlike the lesions of poliomyelitis, are not limited to these sites. It is this diffuse as well as selective distribution that serves to distinguish the lesions from those of poliomyelitis, which
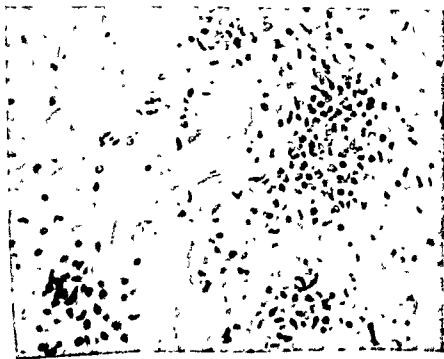
FIG. 14.2—Neuronophagic nodule (right) in ventral gray column of spinal cord. A.F.I.P. 213899. Hematoxylin and eosin stain. X 350.

neuronophagic and cell nodules and with lymphocytic cuffing around nearby blood vessels These lesions are found diffusely throughout the gray matter of the nervous system but are especially concentrated in the ventral columns of the spinal cord cerebellar cortex, diencephalon, mesencephalon, and thalamus The cerebellar pia mater is rather constantly infiltrated with large numbers of lymphocytes

DIAGNOSIS

Only a presumptive diagnosis can be made from the clinical manifestations The disease should be considered in those situations in which swine exhibit fever in addition to signs referable to lesions in the central nervous system The tendency of the disease to spread through a herd especially attacking young animals, at

FIG 14 6—Cell nodules (dark spots) in Purkinje cell and molecular layers of cerebellum A F I P 514663 Nissl s stain X 35
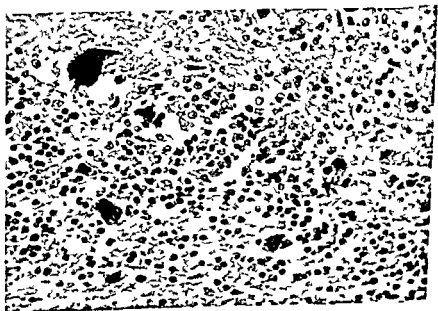
FIG 14 7—Neuronophagic nodule in Purkinje cell layer of cerebellum Note distention and loss of Purkinje cells, only one be ing clearly recognizable A F I P 266606 Hema toxylin and eosin stain X 350

they most closely resemble   These features are also essential in differentiating Teschen disease from hog cholera and African swine fever when nervous symptoms make these diseases diagnostic problems   This point will be discussed further under diagnosis

The lesions in the central nervous system of a pig dying of the infection, or sacrificed during a fully developed stage of the disease, are most widespread in the spinal cord and cerebellum   The ventral horns of gray matter of the cord are most severely affected, although the dorsal horns are not always spared   The intense destruction of nerve cells and the presence of both neuronophagic and cell nodules are characteristic features in the spinal cord (Fig 14 5)   Lymphocytic infiltration of the Virchow Robin spaces is also constant in the involved segments of the cord   Congestion is not unusual and small hemorrhages may be seen   The disease in its early stages affects the cervical cord, but as it progresses, the thoracic, lumbar, and sacral regions become equally involved

The cerebellum is next most severely involved, the lesions being distributed not only in the dentate and roof nuclei but in the cortex and meninges as well   Cell nodules are scattered through the molecular layers, and neuronophagic nodules are present in relation to dead and dying Purkinje cells (Figs 14 6, 14 7)   The meninges over the cerebellum are intensely infiltrated with lymphocytes at the height of the disease (Fig 14 8)

The medulla oblongata and pons are affected similarly to the spinal cord, but usually in a quantitatively less severe manner (Fig 14 9)   Cell nodules and perivascular cuffs are regularly found in most parts of the mesencephalon as well as the diencephalon   The globus pallidus, putamen, caudate nucleus, and claustrum are affected in order of decreasing severity, and usually each is less affected than nuclei in the diencephalon and mesencephalon   The cerebral cortex is also the site of lesions which are moderate in number and rather widely scattered, consisting mostly of small cell nodules and perivascular infiltrations   The peripheral ganglia, including the gasserian, stellate, thoracic sympathetic, and celiac ganglia occasionally undergo neuronal degeneration and lymphocytic infiltration   Similar changes may occur in the spinal ganglia

In summary, the lesions of porcine encephalomyelitis consist of degenerative changes in neurons with formation of

FIG 14 5—Neuronophagic and cell nodules, neuronocrosis, and perivascular cuffing in ventral gray column of spinal cord   A F I P. 514663   Nissl s stain   X 150

guished solely on a clinical basis from those of Teschen disease, although other features of the two former diseases (see Chapters 7 and 8) may be helpful in making tentative separation of these entities Salt poisoning may be presumptively distinguished by the history of excessive salt intake or deprivation of water, the absence of fever, and the failure of the disease to spread by contact Nervous signs of nutritional origin are rare under natural circumstances, fever is not observed, and the disease does not spread by contact Investigation of the feeding practices also may yield clues to the etiology Definitive diagnosis can only be established through laboratory methods

The microscopic lesions in the central nervous system are of particular value in distinguishing Teschen disease from hog cholera and African swine fever From the foregoing parts of this chapter, it will be recalled that neuronal necrosis and neuronophagic and cell nodules are prominent features in porcine encephalomyelitis These are minimal or absent in hog cholera and African swine fever Also, cellular infiltrations occur within the wall of blood vessels in hog cholera and African swine fever, not in the Virchow Robin spaces (see Chapters 7 and 8) These features permit the experienced pathologist to distinguish porcine encephalomyelitis in properly prepared histologic sections Brain and spinal cord should be prepared for histologic study by fixation in adequate quantities of 10 per cent formalin solution The best procedure is to remove brain and cord with aseptic precautions, cut out a few small (1 cm) cubes of tissue from the cerebral hemispheres, brain stem, and cord (for virus isolation), then immerse the remainder of the brain and cord in about ten times their volume of 10 per cent formalin (9 parts water, 1 part formaldehyde solution — containing 40 per cent formaldehyde gas)

Demonstration of the virus should be undertaken only in properly equipped and staffed laboratories Small blocks of brain and spinal cord, collected aseptically and submitted promptly, or frozen at –70° C are used for virus isolation A 10 per cent suspension of these tissues is made in physiological saline solution and inoculated intracerebrally into young swine Recognition of the disease in these inoculated swine is dependent upon the appearance of characteristic symptoms and demonstration of typical lesions in their central nervous system Serologic tests such as complement fixation, neutralization, and immunity tests are not sufficiently developed, at this writing to recommend their routine use

TREATMENT

Treatment of swine affected with Teschen disease is presently based upon nonspecific methods which are rarely successful and are probably ill advised under current conditions in the United States It is highly probable that if the disease should appear in the United States an isolation slaughter method of control would be used and that treatment would not be attempted because of the risk of thereby spreading the infection In Europe, serum from recovered swine has been used in treatment with very little success Some swine have been known to survive following a prolonged course, with careful nursing, but the incidence of residual paralysis is high in such animals Under most circumstances the prognosis is quite unfavorable 90 to 100 per cent of affected pigs die

IMMUNITY

Vaccines have been used in Europe with intermittent success in immunizing swine populations against Teschen disease Vaccine prepared by inactivation of suspensions of infected brain and cord with formalin have been reported to have value in controlling outbreaks of the disease (Hecke, 1955) Patocka *et al* (1955) report some success with a vaccine made from 1 per cent suspensions of spinal

least in enzootic areas, and the appearance of fever, irritability, and convulsions, followed by progressive spinal paralysis are all features which suggest Teschen disease. The diagnosis is currently made on a herd basis, rarely can it be established in an individual animal prior to necropsy.

Manifestations of central nervous disturbance may create diagnostic problems if they appear in the course of hog cholera, African swine fever, salt poisoning, and nutritional deficiency of pantothenic acid. Encephalitic signs in hog cholera and African swine fever cannot be distin-
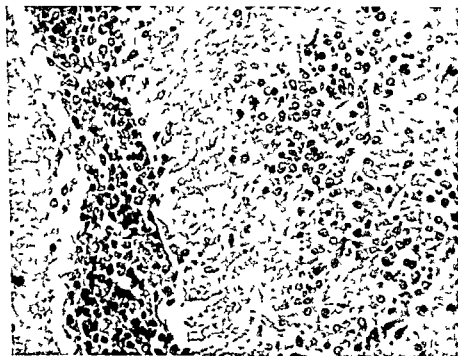
FIG 14 8—Lymphocytes in meninges and cell nodule in molecular layer of cerebellum A F I P 266606 Hematoxylin and eosin stain X 350

FIG 14 9—Cell and neuronophagic nodules in pontile nuclei (dark areas) A F I P 514663 Nissl s stain X 4

HENDRICK, A   1955   Versuche zur Frage einer Vitamin D₂ Prophylaxe bei der Poliomyelitis suum Arch Exper Vet Med 9 736 V B 26–2890

HORSTMANN, D M   1952   Experiments with Teschen Disease (Virus Encephalomyelitis of Swine) Jour Immun 69 379

HUBRIG, Th, AND OHDER, H   1953   Ansteckende Schweinelahme in Thuringen Monatsch f. Vet Med 9 173 V B 24–4056

KAPLAN, M M AND MERANZE, D R   1948   Porcine virus encephalomyelitis and its possible biological relationship to human poliomyelitis Med Vet 43 330

KMENT, A   1940   Zur Histopathologie des Zentralnervensystems bei der Teschener Schweine lahmung Wien tierarztl Monatsche 27 361

KODRUJA, E   1950   Djelovanje krvi svinja hiperimunizranik protiv zarazne uzetosti [The action of blood from hyperimmunized pigs in Teschen Disease] Vet Archiv 20 1 V B 53 2556

——   1952   L apparition et la lutte contre la maladie de Teschen in Slovénie Bull Off int Epiz 38 117, V B 23 1253

KORNYEY, S, AND ELEK, P   1952   Histologische Untersuchungen zur Pathogenese und Patho physiologie der Teschener Krankheit (ansteckende Schweinelahmung) Acta Vet Hung 2 143 V B 23 3075

KUBELKA, V AND PATOCKA, F   1954   Electron microscopy of the porcine encephalomyelitis virus Arch Exper Vet Med 8 666

LARSKI, Z   1955   Hodowla tkankowa wirusa choroby cieszynskiej swin [Tissue culture of Teschen disease virus] Méd vét Varsovie 11 589 V B 26–454

——, AND SZAFLARSKI, J   1955   Szczepionka przeciw chorobie cieszynskiej swin [A vaccine against Teschen disease] Med vet, Varsovie 11 276, V B 26–455

MANUELIDIS, E E, SPRINZ, H, AND HORSTMANN, D M   1954   Pathology of Teschen disease (virus encephalomyelitis of swine) Amer Jour Path 30 567

MAYR, A AND SCHWOBEL, W   1956   Zuchtung des Virus der ansteckenden Schweinelahmung (Teschener Krankheit) in der Gewebekultur Monatsh f prakt Tierheilk 8 49 V B 27–126

MESSOR, C   1956   Lesions in the autonomic nervous system in swine fever and Teschen dis ease   Monatsh f Vet Med II 105

NANI, S, GIGLIETTI, A, AND TIECCO, G   1955   Ulteriori indagini elettroforetiche su carta in sieri di suini trattati con diverse concentrazioni di virus [Paper electrophoresis on the serum of experimentally infected pigs] Boll Ist sieroter Milano 34 318 V B 26 1257

—— SCATOZZA, F AND TIECCO, G   1955a   Ricerche sperimentali sul virus di Teschen I Titolazione del ceppo in studio [Titration] Boll Ist sieroter Milano 34 314 V.B 26 1256

—— —— AND ——   1955b III Comportamento nel fenomeno di emoagglutinazione [Hemagglutination] Boll Ist sieroter Milano 34 327 V B 26 1258

—, TIECCO, G, AND SCATOZZA, F   1955c V Prova di neutralizzazione sul suino con gamma globuline antipoliomieliache di provenienza umana [Action of poliomyelitis immune serum on the virus of Teschen disease] Vet Ital 6 906 V B 26 256

PATOCKA, F, KUBELKA, V AND BOHAC, J   1953   Prispesek k imunoln nakazive obrny vepru (encephalomyelitis enzootica suis) [Immunology of Teschen disease] Ceskoslobensk hyg epid mikrobiol 2 22 V B 25–1016

—— AND SLAVIK, K   1951 Onekterych biolobickych vlastnostech viru encephalomyelitis enzootica suis (Tesinska choroba) [Some biological properties of the virus of encephalo myelitis enzootica suis (Teschen disease)] Vestn csl Akad Zemed 25 461 V B 25– 2276

—— ——, AND BOHAC, J   1952 Onekterych biologickych vlastnostech viru enceph alomyelitis enzootica suis (Tesinska choroba) [Some biological properties of the virus of porcine encephalomyelitis] Ann Acad Tchecosl Agric 25 131 V B 25–109

PEDINI, B, NANI, S, AND GIGLIETTI, A   1955 IV Variazioni del quadro ematomorfologico e glicemico nell infezione sperimentale del suino [Changes in the morphology and sugar level of the blood in experimental infection of pigs] Boll Ist sieroter Milano 34 332 V B 26–1559

PILET, E   1952 La méningo-encéphalomyélite enzootique du porc a Madagascar Bull Off int epiz 38 61 V B 23–2554

ROHRER, H   1954 Das heutige Problem der Schweinepest und Schweinelähme in der Deutschen Demokratischen Republik Monatsh f Vet Med 9 496 V B 25–1020

SCATOZZA F   1955 Ricerche sugli anticorpi neutralizzanti in suini iperimmunizzati verso il virus di Teschen [Neutralizing antibody in pigs hyperimmunized against the virus of Teschen disease] Arch vet Ital 6 45, V B 26–101

SLANINA, L   1955 Porovnavacie studium klinickehopozorovania a pathohistologickych zmien v cns u akutnych chronickych a vyzdravenych pripadov nakazlivei obrny osipanych [Clinical study and histology of lesions in the CNS of pigs with acute and chronic Teschen disease and in convalescents] Vet Cas 4 29, V B 26–835

cord emulsified in liquid paraffin and lanolin   A formalinized aluminum hydroxide adsorbed vaccine has also been described by Larski and Szaflarski (1955) Use of virus cultured in embryonic swine tissues in the preparation of vaccine would appear to have promise but data on this point have not come to the attention of the writer

CONTROL

Methods to control this disease will be different in the Eastern Hemisphere, where the disease is enzootic, from those of the Western Hemisphere, where it has not yet appeared   In Europe the approach seems to be to prevent the spread of the disease by isolation and quarantine and to in

crease the immunity of the swine population in enzootic areas by use of killed virus vaccines

In the United States, control measures are currently aimed at preventing entrance of infected swine into the country and using inspection and quarantine procedures at the borders   Should the disease, despite these measures, appear in the United States, control would depend upon accurate and prompt diagnosis, sacrifice of affected herds, and stringent isolation and quarantine of areas of infection   It is not likely that vaccination would be used as a control measure except as an adjunct to the slaughter and quarantine approach unless this latter method should prove inadequate to eradicate the disease

REFERENCES

(Abstracts in English in the Veterinary Bulletin are indicated after
certain references by V B—followed by volume and abstract number)

BIANCHI E 1953 Il morbo di Teschen (Transmissione sperimentale) Clin Vet Milano 76 193 V B 24 1110

BRAUNER J 1953 Prevencia Proti nakazlivej obrne osipanych podl a novoziskanych poznatkov [Control of porcine encephalomyelitis (Teschen Disease)] Veterinarstvi Brno 3 147 V B 24 2749

— URSINY, AND ZUFFA 1955 Tapasztalatok a bratislavai adszorbealt vakcinaval a fertozo sertesbenulas elleni vedool tasokkal kapcsolatban Javaslat a betegseg eredmenyes elfozta sara Csechoslovakia ban [Results obtained in Czechoslovakia with a Teschen disease vaccine] Mag allator Lapja 10 86 V B 25 2807

— AND —— 1955 Erfahrungen bei der Immunprophylaxe der ansteckenden Schweinelähmung mit Pressburger Adsorbatvakzine und ein Vorschlag zur erfolgreichen Bekämpfung dieser Krankheit in der Tschechoslowakei Arch Exper Vet Med 9 522 V B 26 289

BUCK G AND QUESNEL J J 1954 Sur la paralyse contagieuse du porc (maladie de Teschen) a Madagascar Bull Epiz Dis Afr 2 278 V B 25 2108

DOBBERSTEIN J 1912 Histopathologie des Zentralnervensystems bei der Poliomyelitis des Schweines Zeitschr f Infektionskrankh parasit Krankh u Hyg d Haustiere 59 51

ELEK, P KERTAY (Kerbler) N 1952 Versuche zur Ausarbeitung einer Schutzimpfungs methode gegen die ansteckende Schweinelähmung Acta Vet Hung 1 367 V B 25 1561

FISCHER K AND RÖHRER H 1955 Untersuchungen uber den Wanderungsweg des Virus der Schweinelähmung I Lokalization des Virus im Zentralnervensystem nach intranasaler Infektion Arch Exper Vet Med 9 231 V B 26-1631

— AND STARKE G 1955 Liquoruntersuchungen bei der Poliomyelitis der Schweine Arch Exper Vet Med 5 38 V B 24 1886

FORTNER J 1952 La maladie de Teschen Bull Off int Epiz 38 106 V B 25 1565

GASPARINI G AND NANI S 1955 VI Ricerche sperimentali sul virus di Teschen   Prova di neutralizzazione sul swino con gamma globuline anti Teschen   [Neutralization test in pigs using anti Teschen gamma globulin] Giorn Microbiol 1 120 V B 26 2889

GIUFFITTI G 1955 Il morbo di Teschen Arch Vet Ital 6 425 V B 26-101

— AND FIECCO G 1955 Analisi elettroforetiche su carta in sieri di suini trattati con virus di Teschen [Examination by paper electrophoresis of the serum of pigs artificially infected with the virus of Teschen disease] Arch Vet Ital 6 25, V B 26 102

HECAF, F 1955 Untersuchungen über den Wert von Schutzimpfungen für die Bekämpfung und Tilgung der ansteckenden Schweinelähmung auf Grund einer Analyse des Seuchen geschehens mit statistischen Methoden Wien tierarztl Monatschr 42 606 V B 26 161

— 1956 Comparative statistical examination of the epidemiology of foot and mouth disease Teschen disease and blackleg Zentralbl Bakt I Abt Orig 166 1

Bacterial and Mycotic Infections

SOKOL, A, ROSOCHA, J, AND DALLOS, V   1954   Ucinok ultraziuku na obnovy mozgomiechovy material a imunogenna hodnota ozvucenej adsorbatovej vakciny nakazlivej obrny osipanych [The effect of ultrasonic waves on brain cord material from pigs with Teschen disease and the value of adsorbate vaccine treated with ultrasonic waves] Vet Cas 3 77 V B 25–2001

────   ────   AND SPENIK, M   1953   Immunology, pathogenesis and pathology of porcine encephalomyelitis (Teschen disease)   Vet Cas 2 201

SZAFLARSKI, J   1954   The porcine encephalomyelitis (Teschen disease) research center at Gurnna Poland   Méd vét , Varsovie   10 726

────   1955 Teschen disease in wild pigs in Poland Méd vét , Varsovie 11 20

TIECCO G   1955   La deviazione del complemento in suini, cavie e conigli trattati con virus di Teschen [Complement fixation test in pigs, guinea pigs and rabbits inoculated with the virus of Teschen disease] Arch Vet Ital 6 33, V B 26–103

TREFFNY, L   1930   Massenerkrankungen von Schweinen in Teschner Land   Zverolekarsky Obzor 23 235

TROPA E , AND CORREIA, M A   1954   Encefalomielite em leitões [Encephalomyelitis (Teschen disease) in piglets in Portugal] Rev Cienc Vet , Lisboa 49 369 V B 25–2806

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CHAPTER 15

Listeriosis

Listeriosis is an infectious disease of ani mals and man caused by a bacterial organ ism known under the present classification as *Listeria monocytogenes* It produces an infection characterized by a monocytosis in a number of animal species Murray et al (1926) were the first workers to recover this organism from rabbits and guinea pigs which showed generalized infections with hepatic necrosis In addition to the liver changes and the presence of an exudate in the serous cavities, a marked mononucle osis was observed Because of this great increase in mononuclear cells, the organ ism was designated as *Bacterium monocytog enes* There were no references made to histopathologic changes in the central nervous system

Pirie (1927) working in South Africa on the Tiger River Disease in the ger bille a small native rodent, isolated a similar organism which caused a general ized infection in which focal necrosis of the liver was the most obvious lesion The organism was rather singular and could not be suitably incorporated in the existing bacterial classifications Consequently, he proposed the creation of a new genus in honor of Lord Lister and suggested the name *Listerella hepatolytica* Pirie recog nized the similarity of this organism with that described by Murray et al and pro posed that the name *Listerella monocytog*

enes be used should they prove to be identical Gill (1933) reported a meningo encephalitis of sheep in New Zealand and in 1937 described the organism isolated from the diseased sheep He proposed the name *Listerella ovis* The organism was designated as *Listerella monocytogenes* in the 1934 edition of Bergey s Manual and the disease became commonly known as Listerellosis Later, the name Listeria was advocated by Pirie and accepted by Bergey in the 1948 edition of his Manual of De terminative Bacteriology The disease caused by *Listeria monocytogenes* is now designated as Listeriosis Listeria infection has also been reported in sheep by Hirato et al (1954) in Japan Jungherr (1937) in Connecticut, Biester and Schwarte (1939, 1941) in Iowa Graham et al (1938) in Illinois, and Olafson (1940) in New York Cattle infections have been reported by Jones and Little (1934) in New Jersey, Fincher (1935) and Olafson (1936) in New York Graham et al (1938) in Il linois as well as Biester and Schwarte (1941, 1942) in Iowa Listeria infections in chickens have been reported both in England and the United States Reports from Norway Germany, France, and the United States indicate significant losses in horses were experienced from Listeria in fection Various species of wildlife includ ing foxes, chinchillas, ferrets raccoons,

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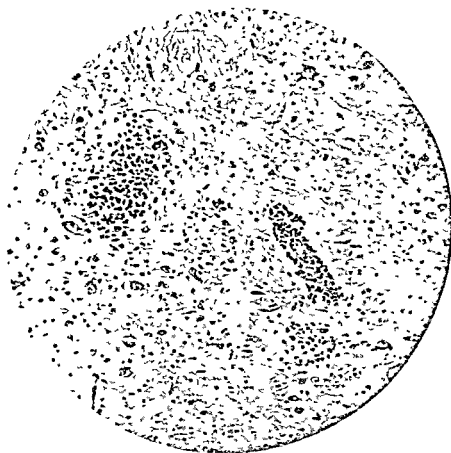
FIG. 15.2 — Swine listerio-
sis, field case. Section of
brain tissue showing a
focal area of infiltration
and perivascular cuffing.
X 150.

known. The extensive geographical dis-
tribution of *Listeria monocytogenes* and
the wide variety of hosts including most
of our domesticated animals, numerous
species of wildlife as well as man indicate
the existence of many reservoirs of infec-
tion and numerous vectors which could be
responsible for the spread of the disease.
The incidence of listeriosis as a primary
infection in swine is probably more preva-
lent than generally believed because swine
seem to be far more resistant to fatal in-
fections than some of the other domesti
cated animals.

Listeria monocytogenes has been isolated
from many species of animals which have
contracted serious or fatal infections with
other pathologic agents. Hog cholera, pox,
infectious enteritis, pneumonias, salmonel-
losis, and brucellosis are examples of pri-
mary infections from which the Listeria
organism has been isolated. The carrier
state reported in healthy sheep by Shimizu
et al. (1954) indicates that the organism
may be present in carriers of many species

including man, without any clinical mani
festation of the disease. It appears that
any time the natural resistance of any
animal with a latent infection or in a car-
rier state is sufficiently reduced from any
cause whatsoever, listeriosis may develop
and contribute to the fatal termination of
other primary infections.

Experiences recorded under experimental
conditions give us the only information
available on the transmission of listeriosis.
These hardly seem applicable to natural
infection. Intravenous and intracranial
inoculation of swine with pure cultures or
with infected material have produced
rapidly fatal cases of listeriosis. Subcu-
taneous, intramuscular, intraperitoneal, in-
tranasal, and oral experimental infections
in some instances produced rather mild re-
actions followed by recovery, especially in
suckling or weanling pigs; but in the more
mature animals, little if any reaction was
recorded. The means of transmission of
listeriosis by natural infection has still to
be determined.

voles, and skunks have been found infected with the Listeria organism A number of human infections have been reported from the United States and Europe

Slabospickij (1938) appears to be the first to report Listeria infection in swine He isolated the organism from young infected pigs raised on a Russian farm and designated the organism as *Listerella suis* Subsequent European reports of listeriosis in swine include those of DeBlieck and Jansen (1942) and Solomkin (1954) The greatest number of reports of swine listeriosis in the United States has come from the north central states where the swine population is the greatest. Reports by Biester and Schwarte (1940), Rhoades and Sutherland (1948), Helmbolt *et al* (1951), Gray *et al* (1951), and Ryu (1955) indicate that listeriosis in swine is not uncommon in this country

ETIOLOGY

Listeriosis is caused by a small bacterial organism known as *Listeria monocytogenes* These bacteria occur as rods 1–2μ in length and approximately 0 5μ in diameter On culture media, filaments as long as 4μ may be observed The organisms may be seen singly, in pairs, or in short chains Polar flagella which stain with difficulty are responsible for its motility Young cultures usually exhibit the greatest activity They do not form capsules or spores The organisms stain readily with practically all the aniline dyes and are Gram positive Older cultures frequently show bipolar staining They are not acid fast.

L monocytogenes is aerobic and facultative, it grows well on any of the media used for the cultivation of pathogenic bacteria Some laboratories prefer blood agar plates on which this organism produces pinpoint deep colonies and flat, bluish white surface growths The deeper colonies are surrounded by narrow hemolytic zones Minute colonies surrounding the inoculation area develop on semisolid media A slight turbidity develops in broth with a granular sediment accumulating in the bottom of the tubes The optimum temperature appears to be about 37° C.

but the organisms will grow between 20° and 40° C The culture media should be adjusted to pH 7 0–7 2

Acid without gas is produced from salicin, rhamnose, and dextrose in 24 hours following inoculation Acid is produced in lactose, glycerol, sucrose, dextrin, and maltose in 7 to 10 days Fermentative changes are rather slow and variable in the presence of xylose, sorbitol, trehalose,

FIG 15 1 – Listeria monocytogenes isolated from pig X 990

and galactose Certain strains of Listeria may act on levulose No fermentation reactions have been recorded for arabinose, inulin, inositol, dulcitol, and mannitol The organism does not reduce nitrates form H$_2$S, or produce indol Gelatin is not liquefied Litmus milk supports growth but is not coagulated Some strains of the organism produce slight acid formation

Listeria monocytogenes is capable of producing disease through natural infection in sheep, cattle, swine, guinea pigs, rabbits, chickens and man as well as many species of wildlife including rodents, foxes, and skunks

TRANSMISSION AND DISTRIBUTION

The mode of transmission and the incubation period by natural infection is not

actions (107.4° to 107.7° F.) and in 24 hours were unable to stand. Clinical signs of a septicemia developed. The pigs died on the second and third day following inoculation. The swine inoculated intraperitoneally developed elevated temperatures (105.7° to 105.9° F.) for a day or two. There were no symptoms observed for two weeks following inoculation. There was no reaction following subcutaneous inoculation of swine. The intranasal inoculations produced elevated temperatures (105.3° to 105.4° F.). A cough developed two days following inoculation. Spasms were reported later during the four days following inoculation. The pigs made an uneventful recovery. The orally infected pigs showed no elevated temperature reactions or clinical signs.

Field cases involving swine from 40 to 150 lb. exhibited varying degrees of severity in their reactions to the disease. Some individuals developed incoordination or a partial posterior paralysis. The forelegs were characterized by an accentuated stilted gait. The swine appeared nervous and were easily excited. The clinical manifestations in the larger swine might be confused with a number of other diseases including nutritional disturbances. A diagnosis cannot be made from clinical symptoms manifested by the infected swine.

COURSE

In swine listeriosis which assumes a septicemic form or produces symptoms of a severe central nervous disorder runs a rapid and fatal course. Under field conditions the infected animals seldom survive for more than four days after the manifestation of well-defined clinical signs. Under experimental conditions the swine inoculated intravenously or intracranially develop elevated temperature reactions and clinical signs within 24 hours and in the majority of cases die or become moribund in 48 to 72 hours. Experimental swine inoculated intraperitoneally, intramuscularly, intranasally, or orally may or may not show moderate temperature elevations or mild and transient clinical signs followed by recovery.

PATHOLOGY

Necropsy examination of field cases shows no significant changes that are suggestive of the nature of the disease. Histopathological studies of the central nervous system disclose a definite meningitis characterized by a severe monocytic infiltration. Numerous blood vessels, particularly those in the area of the pons, reveal perivascular cuffing. Many foci of monocytic infiltration are found. A considerable number of polynuclear cells are present in some areas. An increased number of monocytes are present in the circulating blood.

Experimentally inoculated swine are more likely to reveal the true nature of the tissue changes caused by Listeria infection than field cases, in which concurrent infections often complicate the picture. Pigs inoculated intracranially with pure cultures of this organism die about 24 hours later. Pure cultures may be recovered from the brain tissue. Negative results are obtained from the bacteriological examinations made of the parenchymatous organs. Severe monocytic infiltrative meningitis, perivascular cuffings, and focal infiltrations of monocytes are observed in the histopathological studies of the brain tissues. The tissues surrounding the ventricles and pons contain the most advanced cellular changes, but characteristic changes are found in other parts of the brain and spinal cord. The lining cells of the neural canal and ventricles are destroyed in many places and monocytic infiltrations appear in the deeper structures. The kidneys are slightly swollen, but no gross diagnostic changes can be detected. The histopathology reveals focal monocytic infiltrations which oftentimes involve the glomeruli and adjoining tissue. The liver is also swollen. It does not have the yellowish color often observed in field cases of ovine listeriosis which usually run a less acute course. Considerable congestion and definite monocytic infiltration are observed in the microscopic examination of the tissues.

Ryu (1955) reported the isolation of *Listeria monocytogenes* from a field case

CLINICAL SIGNS

The clinical manifestations in swine as the result of Listeria infection vary considerably according to reports found in the literature. Concurrent infections undoubtedly exert definite effects on the clinical signs manifested, according to some reports. Biester and Schwarte (1910) observed several outbreaks of listeriosis in swine which occurred in Iowa. The incidence of the disease in young swine was much greater than in the older individuals in the same herds. Considerable ranges of temperature elevation were observed. Infected swine presented clinical signs of a central nervous disorder. The majority of the larger swine manifested various degrees of shaking or trembling. Some individuals dragged their hind legs or showed various degrees of incoordination while the movements of the forelegs in many instances became a characteristic stilted gait similar to that observed in tetanus. Under experimental conditions 50-lb. pigs which were inoculated intracerebrally with pure cultures of swine origin showed a severe central nervous reaction and died in 24 hours. Repeated intramuscular inoculation of pigs with swine strains failed to cause any clinical signs in animals which were kept under observation for two months Listeria strains of ovine origin on repeated intramuscular inoculations produced no clinical reactions for a period of one month while the inoculations were being made; however, about one month after the inoculations were discontinued, the pigs developed clinical signs of a central nervous disorder. The day following the appearance of clinical signs the pigs were unable to stand. Pure cultures, injected intravenously and fed to pigs ranging from 160 to 170 lb. in weight, failed to produce any untoward effects.

Ryu (1955) isolated Listeria from swine which apparently were infected with other pathogenic organisms. These cultures were inoculated into suckling pigs up to two months of age under experimental conditions. Following intravenous inoculation the pigs showed elevated temperature re-
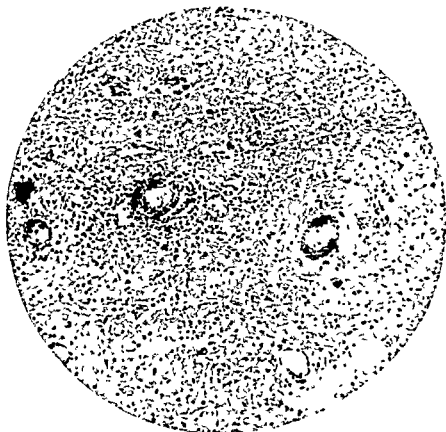
FIG. 15.3 — Swine listeriosis, field case. Section of brain tissue showing leukocytic infiltration and perivascular cuffing X 150.

severe disturbances of the central nervous system, similar to many field cases in sheep and cattle Symptoms of a central nervous disturbance with various degrees of incoordination and progressive weakness followed by death which is typical of most neurotropic infections is characteristic of listeriosis of the younger swine Older animals seem to be more resistant as the symptoms are less severe and recoveries are not uncommon *Listeria monocytogenes* is recovered from the brain and cord tissues and not from the blood or internal organs such as the liver, spleen, and kidneys The histopathology of the central nervous tissue includes meningitis, perivascular cuffing, focal infiltration, and an increase of monocytic cells in the blood

The organisms are apparently confined to the foci of infection in the tissues of the brain and cord Attempts to isolate the organism by inserting a sterile platinum loop into the brain tissue and inoculating culture media terminates in negative results in more than 60 per cent of the

cases, according to Biester and Schwarte (1940) Trituration of composite samples of brain and cord in a mortar and inoculating the brain emulsion into laboratory animals and culture media result in a high percentage of positive culture isolations A 10 per cent tissue emulsion agitated in a mechanical shaking device for 15 to 20 minutes will achieve the same objective Electric blenders will thoroughly emulsify infected tissue in 5 minutes, breaking up the foci of infection and uniformly distributing the organisms throughout the emulsified tissue For making isolations this is the method of choice

A method of verification of a clinical diagnosis of listeriosis when initial isolation by culture was negative was demonstrated by Gray *et al* (1948) in a refrigeration storage technique They showed that the chances for successful isolation of the organism were enhanced by storing the infected brain at 4° C for a period of several weeks prior to reculture

The literature contains a number of re

FIG 15 5 — Swine listeriosis experimental case Section of brain tissue showing meningitis X 150
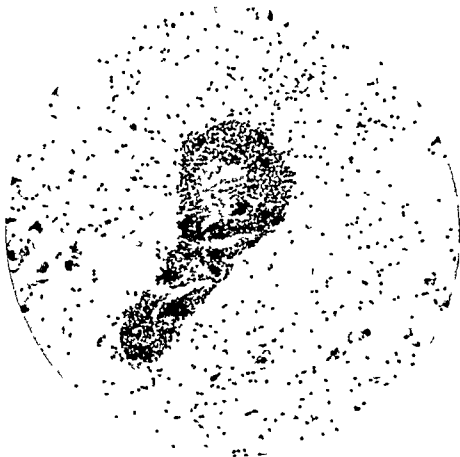
FIG 15 4 — Swine listerio sis, experimental case Section of brain tissue showing perivascular cuffing   X 150

diagnosed as pleuropneumonia in a two month-old Hampshire pig No nervous symptoms or clinical manifestations of an encephalitis were observed Listeria organ isms were isolated from the liver, spleen, and kidneys Pasteurella organisms were isolated from pneumonic areas of the lungs In another field case involving a septicemia in a pig from which organisms of the Pasteurella and Erysipelothrix groups were isolated from the brain and lungs, Listeria organisms were also isolated from brain cultures Cultures made from the liver, spleen, and kidneys were negative Kerlin and Graham (1945) isolated Lis teria from grayish focal liver lesions of an unthrifty pig in which no symptoms of encephalitis were observed Rhoades and Sutherland (1948) reported a case of con current infection with hog cholera and *Listeria monocytogenes* in a 4 month-old pig Listeria organisms were isolated from the liver, spleen, and kidney in pure cul ture No symptoms of encephalitis were observed The lesions described were typi

cal of hog cholera and the Listeria infec tion was considered secondary to hog cholera

The numerous reports of listeriosis in swine, especially with concurrent infec tions, produce confusing pathologic pic tures to the experienced pathologist, as well as the veterinarian The lesions found in various organs of the body in field cases complicated by secondary infections differ considerably from tissue changes ob served in experimental transmission stud ies Consequently, it is necessary to cor relate the isolation and identification of the bacterial organism with the pathologic changes in evaluating the lesions when secondary infections are involved

DIAGNOSIS

The number of studied and recorded field cases of Listeria infection in swine is rather limited and no generalized con clusions can be formulated As it occurs in the field as well as in fatal experimental infections, the disease is manifested by

BIESTER, H E, AND SCHWARTE, L H 1939 Studies on Listerella infection in sheep Jour Infect Dis 64 135

——— AND ——— 1940 Listeriosis in swine and criteria for diagnosis Proc 44th Ann Meet U S Livestock San Assn, p 42

——— AND ——— 1941 Bovine Listerellosis in Iowa with studies on a recovered case No Amer Vet 22 729

BOLIN, F M TURN, J, RICHARDS, S H, AND EVELETH D F 1955 Listeriosis of a skunk No Dak Agr Exp Sta, Bimo Bull 18 49

DeBLIECK L AND JANSEN, J 1912 Listerellose bij biggen Tijdschr v Diergeneesk 69 575

FINCHER, M G 1935 Meningo encephalitis of ruminants Cornell Vet 25 61

GILL, D A 1933 Circling disease a meningo encephalitis of sheep in New Zealand Vet Jour 89 258

——— 1937 Ovine bacterial encephalitis (circling disease) and the bacterial genus Listerella Australian Vet. Jour 13 46

GRAHAM R DUNLAP, G L, AND BRANDLY, C A 1938 Ovine and bovine listerellosis in Illinois Science 88 71

GRAY, M L STAFSETH, H J AND THORP F JR 1951 A four year study of listeriosis in Mich igan Jour Amer Vet Med Assn 118 242

——— ———, SHOLL, L B, AND RILEY, W F JR 1948 A new technique for isolating listerellae from the bovine brain Jour Bact 55 471

HELMBOLT, C F JACOBS R E AND CASE L I 1951 An outbreak of porcine listeriosis Vet Med 96 347

HIRATO K K, SHIMIZU, T, ONO, S SATO Y, YAWATA Y AND NISHIHARA Y 1954 Bacteriological observations on an outbreak of ovine listeriosis in Sapporo Jap Jour Vet Res (Hokkaido University) 1 191

JONES F S AND LITTLE, R B 1934 Sporadic encephalitis in cows Arch Path 18 580

JUNGHERR, E 1937 Ovine encephalomyelitis associated with Listerella infection Jour Amer Vet Med Assn 91 73

KERLIN D I, AND GRAHAM R 1945 Studies on Listerellosis VI Isolation of *Listerella mono cytogenes* from the liver of a pig Proc Soc Exper Biol and Med 58 351

MURRAY, E G D, WEBB, R A, AND SWANN, M B R 1926 A disease of rabbits characterized by a large mononuclear leucocytosis caused by a hitherto undescribed bacillus *Bacterium monocytogenes* (n sp) Jour Path and Bact 29 407

OLAFSON, P 1936 *In* The Practice of Veterinary Medicine, 2nd ed D H Udall Ithaca N Y p 113

——— 1940 Listerella encephalitis (circling disease) of sheep cattle and goats Cornell Vet 30 141

PIRIE J H H 1927 A new disease of veld rodents Tiger River Disease So Afr Inst Med Res 3 163

RHOADES, H E AND SUTHERLAND A K 1948 Concurrent *Listerella monocytogenes* and hog cholera infections Jour Amer Vet Med Assn 112 451

RYU, E 1955 Three cases of listeria infection in swine. Mem Coll Agr Nat Taiwan Univ 4 1

SCHWARTE, L H, AND BIESTER, H E 1942 Listerella infection in cattle Amer Jour Vet Res 3 165

SHIMIZU, K OTSUKA G, AND OKA M 1954 Guanofuracin media for isolation of *L monocy togenes* and its practical application Jap Jour Vet Res 2 1

SEELIGER, H 1955 Listeriose Beitrage zur Hyg u Epidem 8 (143 pages with references)

SLABOSPICKIJ, T P 1938 Pro novni mikroorganizm vidilenii vid porosyat Nauk Zap Kiev vet Inst 1 59

SOLOMKIN, P S 1954 Listerella infection in swine Veterinariya Moscow 31 24

ports in which Listeria infection occurs concurrently with other conditions such as pneumonias septicemias erysipelas, hog cholera and other diseases The lesions described in these cases usually are typical of those associated with the primary in fection Listeriosis under these conditions may be considered as a secondary infec tion Listeria monocytogenes has been iso lated from the blood and most of the in ternal organs of swine which have de veloped secondary infections The wide geographical distribution of Listeria and the number of animals and birds both wild and domestic serving as vectors and foci of infection make it possible for the organism to be harbored in animals and man without any detrimental effect until the resistance of the host is reduced suf ficiently by a primary infection or other unfavorable conditions The organism may then become active causing a primary infection or assume a role secondary to a primary condition Shimizu et al (1954) detected carriers of the Listeria organism among healthy sheep

It is important that a differential diag nosis be made from Aujezky's disease, rabies, encephalitis, meningitis and other diseases exhibiting symptoms of a central nervous disturbance Furthermore Listeria encephalitis of swine is distinct from a clinically recognized field condition desig nated as shivering pigs The latter is generally believed to be of hereditary ori gin Culture media of various types have been used in attempted isolation of bac terial organisms from the brain liver, kid ney and spleen of so-called shivering pigs with negative results Histopathological studies of the brain and cord tissues failed to show tissue changes similar to those as sociated with listeriosis

Since the conditions produced by pri mary infections of Listeria monocytogenes in swine may be confused symptomatically with a considerable number of diseases,

the only certain method of a positive diag nosis is the isolation and identification of the organism Various serological tests commonly used for diagnostic purposes have been inconsistent

TREATMENT

Various sulfa derivatives used alone and in conjunction with antibiotics have shown beneficial results in arresting the course of the disease in experimental animals Peni cillin Chloramphenicol Aureomycin Terramycin and various other antibiotics with or without streptomycin have also been used with some degree of success under experimental conditions (Seeliger, 1955) Repeated treatments in order to maintain certain optimum concentrations of these therapeutic agents in the blood stream for an extended period of time seem necessary for effective treatment. The effectiveness of this type of treatment in swine has not been determined Such treatment involving multiple or repeated administration of therapeutic agents would be economically prohibitive in vet erinary practice A single large dose might have a favorable effect on the course of field infections in the larger swine es pecially in cases where clinical symptoms are mild Suckling pigs showing severe symptoms of a central nervous disturbance usually do not respond to treatment.

IMMUNITY

There is no information available on the immunity produced by Listeria infec tion in swine Immunization studies con ducted on sheep indicated that only about half of the sheep in the flocks vaccinated were protected from challenge inoculations of broth cultures in experimental studies. The incidence of listeriosis in swine is not prevalent enough in this country to war rant herd vaccination even if a suitable method of immunization were developed

REFERENCES

Bergey D H 1931 Manual of Determinative Bacteriology Williams and Wilkins Co Balti more p 328

———— 1948 Manual of Determinative Bacteriology Williams and Wilkins Co., Baltimore P 408.

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CHAPTER 16

Leptospirosis*

Leptospirosis has been recognized as an important disease of swine only since about 1950 At a meeting of research workers in 1952 the importance of leptospirosis was discussed, and the consensus appeared to be that although the infection is prevalent in swine as indicated by the presence of antibodies, nevertheless the infection produced essentially an inapparent disease Since that time, observations by veterinary practitioners and reports from a number of research laboratories have confirmed the widespread incidence of leptospirosis in swine and have also established the importance of the economic losses from the disease

The U S D A report of losses from live stock diseases for 1954 included an estimated loss in baby pigs of $138,000 000 There are probably several diseases which contribute to this loss, but leptospirosis appears to be of increasing importance in this category It is true that the disease may be present in a herd of swine and

produce no evident disease but on the other hand when it is introduced into a breeding herd the loss of stillborn pigs and squealers which die within the first week after birth may approach 100 per cent of the pig crop for the season

ETIOLOGY

The most common cause of leptospirosis in swine in the United States is *Leptospira pomona* This species was first isolated in Australia by Clayton *et al* (1937) from a person suffering with what was called 'seven day fever' This person had been drinking creek water prior to appearance of the disease Subsequently the same organism was demonstrated in swine and cattle

L pomona is a spiral shaped slender rod which measures about 0 1 to 0 2μ in diameter and 3 to 10μ in length The organism is actively motile It can be observed microscopically by means of dark field illumination but cannot be seen with regular lighting except in preparations using a silver impregnation method of staining None of the commonly used stains assist in making the leptospiras visible Morphologically *L pomona* possesses no outstanding characteristics which aid in differentiating it from other species of the genus

The leptospiras also differ from other bacteria in their cultural characteristics They grow only in special media which

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which are present in the blood and other organs Some of the leptospiras pass from the tubules with the urine into the bladder and in animals in which the pH and other characteristics of the urine are satisfactory, the viable organisms are voided in the urine Such animals are called carriers or 'urinary shedders and they are the important reservoir of infection for susceptible animals Thus a cycle of infection is readily established in which the carriers in a herd maintain a source of infection for young animals on the premises and for susceptible animals which may be added to the herd The swine carriers may continue to shed viable leptospiras in the urine for six months or more

Leptospiras in the urine of swine remain viable for at least several hours if the urine is neutral or slightly alkaline (Ferguson et al , 1956) Since the organisms will not withstand drying, their viability depends upon the deposition of urine in a moist place Transmission may be direct from the infected urine coming into contact with the susceptible animal by way of splashing in the eyes nose, mouth, or abrasions of the skin Indirect transmission depends on the environment of the animals When the infected urine is deposited in a poorly drained area, the organisms may survive for at least a few hours Gillespie et al (1957) reported that in certain types of water L pomona apparently survived for 10 days

Infected boars have been definitely incriminated in introducing L pomona into a herd of breeding sows, however, the author knows of no proven demonstration of transmission by way of coitus Ferguson and Powers (1956) did demonstrate that a culture of L pomona introduced into the vagina immediately following breeding produced a typical leptospirosis in all of the six gilts exposed Thus, it would appear that if L pomona is present in the semen of a boar, he could readily infect the sow at coitus

The observations of Ferguson et al (1956) suggest that the transmission from infected boars to sows was indirect rather than direct Several sows and gilts mated

to two infected boars were negative serologically at 2 to 3 months of pregnancy but subsequently became infected from contact with other infected sows It was presumed that one or more of the gilts mated with the infected boars were exposed to infected urine and that subsequently such infected sows became urinary shedders and served as a source of infection to the other susceptible swine

Morter and Morse (1956) have demonstrated that L pomona can spread from swine to cattle or from cattle to swine by way of infected urine Although differences in virulence of various strains of L pomona have been demonstrated there is little or no evidence that these strains have any specificity for a given species of animal Bryan (1957a) failed to demonstrate differences in immunity in swine between those given a bacterin of bovine origin and those given a bacterin derived from a strain isolated from swine

DIAGNOSIS

Leptospirosis had undoubtedly been occurring in swine for many years prior to its recognition This was, in large part, due to the difficulties involved in demonstrating the causative organism

Leptospiras will not grow in any of the conventional culture media but require a special medium containing approximately 10 per cent serum and with the pH adjusted accurately at 7 to 7 2 At primary isolation the leptospiras grow slowly and may reach a detectable level only after 5 to 30 days of incubation at 30° C

In addition to the special requirements for cultivation, the leptospiras can be detected microscopically only by special methods These organisms are not made visible by any of the common bacteriological stains Methods involving silver impregnation can be used for demonstrating leptospiras in tissue (see section on histopathology) The best procedure for microscopic examination is the use of dark field illumination The optical system which has given best results includes a research type microscope with a substage dark field condenser in place of the usual

contain about 10 per cent normal serum A modified Schuffner's medium (Kelser and Schoening 1948) and Chang's medium (Chang, 1947, Morse et al, 1955) are probably most commonly used for the cultivation of leptospiras The organisms do not ferment carbohydrates nor are there other physiological characteristics which aid in their identification Even though the leptospiras multiply more rapidly at 37°C their death and degeneration also occur more rapidly, so for optimum growth temperatures of 25° to 30°C are generally used for incubation The organisms grow relatively slowly, with a maximum concentration in a well balanced medium being attained between 3 and 7 days

The pH of the medium, as well as other environmental factors, is very important for the survival of leptospiras Optimal conditions for growth and survival seem to be in a narrow range around pH 7 0 Above pH 7 4 growth is inhibited Below pH 6 2 the rate of growth is reduced and, in fact in this slightly acid medium the organisms die and undergo autolysis

INCIDENCE

Leptospirosis is much more prevalent in swine than is commonly suspected, principally because the disease caused by L pomona may assume an inapparent form L icterohemorrhagiae was demonstrated in Europe in swine ill with an unusual disease (Klarenbeek and Winsser, 1937, Field and Sellers, 1951, Nisbett 1951) Gsell (1946) reported that 13 per cent of apparently normal swine in Switzerland had antibodies for L icterohemorrhagiae Infection of swine with this species also has been observed in the United States (Bohl and Ferguson, 1952)

L pomona is the most common cause of the disease in swine in the United States, and this may be true in other parts of the world as well Mochtar (1940) in Asia and Savino and Rennella (1945) in South America reported the isolation of L pomona from the kidneys of apparently healthy swine The first reported isolations of this species of Leptospira in the United

States was that of Gochenour et al (1952) Bryan et al (1953) and Bohl et al (1954), in the United States, and Ryley and Simmons (1954), in Australia, have reported more recently on the isolation of L pomona from aborted swine fetuses

The results of serological tests with L pomona from various areas of the United States indicate that swine leptospirosis is present throughout the country at levels as high as 20 per cent Reports from Europe, South America, and Australia reveal somewhat comparable levels of prevalence, suggesting that the disease is of world wide significance

TRANSMISSION

Leptospiras have a particular affinity for the kidneys of the infected animal, and this characteristic is especially important in the transmission of the organisms from animal to animal Regardless of the species of Leptospira or the species of animal involved, the disease pattern is essentially the same, with variations in the severity of the symptoms and lesions produced

The leptospiras enter the body through breaks in the skin, through the mucous membranes, by way of the conjunctiva, or in other words, through any means by which the organism can gain access to the tissues In the susceptible animal very few leptospiras are required to establish an infection The organisms invade the circulatory system where they multiply and within 2 to 7 days may be demonstrated in all of the visceral organs as well as the blood

Within 5 to 10 days antibodies for the leptospiras may be detected in the blood serum and, at this time, the organisms can no longer be readily demonstrated in the peripheral blood The leptospiras are very sensitive to the specific antibodies, first agglutinating and then disintegrating promptly, presumably due to lysis Complement is not required in this bacteriolytic process

After the appearance of antibodies the leptospiras can be demonstrated in the kidneys where they appear to be localized in the tubules In this area the organisms reproduce, protected from the antibodies

Stoenner (1954) described a rapid plate agglutination test and capillary tube test both of which were designed to simplify the serologic diagnosis of leptospirosis Antigen for the plate agglutination test is now available commercially with directions for its proper use The test is very sensitive, and a positive reaction is not nearly so evident as that seen in the brucella or pullorum plate tests It is important that the operator follow the directions carefully and include known negative and positive sera to insure an accurate interpretation of the results

Bryan (1957b) reported a modification of Stoenner's method which included treatment of suspensions of L pomona with Giemsa stain This gave an antigen which, in the presence of antibodies, produced a much clearer positive reaction

ACUTE LEPTOSPIROSIS

Only a small percentage of the swine infected with L pomona develop clinical evidence of the disease (Ryley and Simmons, 1954, Ferguson et al, 1956 Morse et al, 1958) In many, if not most, of the naturally occurring cases the caretaker does not recognize the presence of illness particularly in a large herd Characteristically the disease spreads from animal to animal in the herd so that possibly only one or a few will be in the acute phase of the disease at a given time These animals may show various levels of inappetence fever, and diarrhea, but these usually persist for 1 to 3 days and may be easily overlooked Careful observation of experimentally exposed swine has revealed the appearance of illness, but even in these cases the signs are transient and mild

The occurrence of hemoglobinuria in a gilt with a febrile reaction and inappetence was reported by Ferguson et al (1956) This occurred in a naturally occurring outbreak of the disease There have been few other reports of hemoglobinuria in swine leptospirosis which suggests that this severe form of the disease is rare

Ferguson and Powers (1956) reported on the changes in the leukocyte count in swine experimentally infected with L pomona There was a trend toward an increased number of leukocytes during the fourth to eighth days after exposure This leukocytosis appeared to be an absolute increase in the number of neutrophils many of which were immature forms

It can be concluded, then, that the leptospirosis in swine caused by L pomona produces only a mild form of the disease which is usually inapparent Sanger (1957) has reported that there is little gross or microscopic pathological change evident in swine killed during the acute phase of leptospirosis

The principal lesions of swine leptospirosis accompany the chronic form of the disease, which is characterized by localization in the kidneys The chief economic loss also appears during the chronic phase in the form of abortion or the birth of weak pigs which fail to survive

CHRONIC LEPTOSPIROSIS

Although there are scattered lesions in the kidneys of swine in the chronic form of leptospirosis, the disease is ordinarily present without any apparent manifestations The disease is self limiting, and recovery with complete elimination of the leptospiras from the kidney usually occurs within 6 months after the initial infection Abortion occurs during this period, usually during the last 3 weeks of the expected gestation

The lesions described in the following section are those one will see at 1 to 3 months following infection The gross lesions are confined to the kidneys, which are pale in color and show a variable number of small grayish foci over the entire surface as well as on cut section Some of the foci are slightly elevated above the surface There are no adhesions between the capsule and the cortex Similar lesions were described in bovine kidneys by Hadlow and Stoenner (1955) and Mathews (1916)

Microscopically, lesions are present in

Abbe condenser A strong light source is reflected into the condenser with the conventional mirror A 15X ocular and a 10X objective, giving a magnification of 150X, give a good result for reading the agglutination lysis test and for the examination of urine in drops on an open slide preparation For detailed examination of the organisms in tissue, blood, or cultures, a cover slip preparation is examined under a 40X objective with either a 10X or 15X ocular

Experience is required in examining known leptospiras before attempting to recognize the organisms in tissue or body fluids Schirren (1953) attempted to explain the development of the bodies commonly called pseudospirochetes These bodies which closely resemble leptospiras especially when viewed by the uninitiated, develop in preparations containing erythrocytes Hypertonicity, which may result from evaporation from a prepared slide and a slight amount of heat will cause the extrusion of filamentous bodies Some of these may become detached from the erythrocyte, and Brownian motion closely simulates the movements of living bodies Photomicrographs of such bodies, prepared by Schirren (1953), indicate the similarity to leptospiras

Laboratory animals, chiefly hamsters and guinea pigs, can be effectively used in demonstrating leptospiras from blood, tissues, or urine of animals suspected of being infected Generally ½–2 ml of inoculum are introduced into the peritoneal cavity If *L pomona* is present in the inoculum, the animals will show evidence of infection by the fourth or fifth day, usually by a rise in body temperature Blood, collected aseptically from the heart on the fourth or fifth day, is placed in the special culture medium in amounts of 0 05 to 0 5 ml *L pomona* can usually be obtained in pure culture, even from heavily contaminated urine or tissue, by this method

Although the cultural method is essential for final confirmation of the presence of leptospirosis and identification of the

the species in a herd of swine, the procedures are not practical for routine diagnosis of the disease in individual animals The serological tests fill a very important role in the recognition of leptospirosis The agglutination lysis test is generally accepted as the most reliable for demonstrating antibodies for the various species of Leptospira

AGGLUTINATION-LYSIS TEST

Living cultures in liquid medium are used as the antigen Generally, cultures which have incubated 3 to 7 days at 30°C have been used, but there is need for standardization of the density Stoenner (1955) referred to the extensive variation in interpretation of test results when the concentration of leptospiras in the antigen is uncontrolled A serum might appear to be negative (a titer of less than 1 100) with a dense antigen, while with a less concentrated preparation the serum might show agglutination in a titer of 1 400 The workers in this field have been attempting to develop methods by which the concentration of organisms can be measured accurately Some standardization, of not only the antigen but other steps in the procedure, is needed to improve the repeatability of the test in the various laboratories A committee was appointed at the 1957 meeting of the American Association of Veterinary Bacteriologists, charged with the responsibility of establishing procedures which may lead to such standardization

The serum to be tested for antibodies is diluted, for example 1 5, 1 50, 1 500, and 1 5000, then 0 1 ml of each dilution is placed in a small, chemically clean test tube An equal volume of the living culture of leptospira is added to each dilution and a control tube containing saline and culture is included in each test The tubes are shaken to mix the contents of each tube thoroughly After standing at room temperature (20 to 25°C) for 30 minutes to 2 hours, the tests are examined under dark field illumination for evidence of agglutination and lysis

of the chronic form of the disease at the time of examination might well influence this occurrence

An occasional petechial hemorrhage is seen in the interstitial spaces Infrequently a few erythrocytes can be found in a tubule Congestion of vessels is not apparent.

Glomerular changes are both frequent and severe Some glomeruli are swollen completely filling Bowman's capsule, and adhesions may form between the capillary loops and the parietal layer of Bowman's capsule (Fig 16 3) Monocytes, neutrophils, and lymphocytes are present in some glomeruli (Fig 16 4). Other glomeruli are atrophic and fragmented some are shrunken, dense, and floating free in Bowman's capsule (Fig 16 5), while some have disappeared entirely Others, as shown by Masson's trichrome stain (Lillie, 1954), are undergoing fibrosis with thickening of Bowman's capsule, obliteration of the capsular space, and complete loss of separation between the capsule and glomerular tuft (Fig 16 6) Almost without exception, where Bowman's space is evident, it contains eosinophilic granular detritus and some contain erythrocytes Langham et al (1958) described essentially the same changes

In silver stained sections (Lillie, 1954) of the kidney of a pig killed 30 days after infection, nearly every tubule in a high power field contained leptospira, but the estimated average infection in all tubules for an entire section of the cortex (1 cm x 1 cm) was about 50 per cent The greater number of infected tubules was in the cortex with diminishing numbers in the medulla

As nearly as could be determined morphologically in silver stained sections, leptospiras were present in all segments of the nephron unit except the renal corpuscle Infection extended from the proximal convoluted tubules immediately beneath the capsule, completely across the width of the kidney, to the last row of collecting tubules at the tip of the papillae

Leptospiras in silver stained sections appear as minute, black threadlike structures which are present singly or in such concentrations that they form black opaque

FIG 16 3 — Note adhesions between the capillary tuft and parietal layer of Bowman's capsule at two places (arrows) with much debris in Bowman's space Vascular pole up, urinary pole down Trichrome X 550

the tubules, glomeruli, and interstitial spaces and are both inflammatory and degenerative in nature.

The grayish foci are caused by the infiltration of inflammatory cells. These foci are found immediately beneath the capsule, throughout the cortex, and in the medulla. The predominant cell is the lymphocyte; however, monocytes and neutrophils are usually present (Fig. 16.1). In occasional animals there is a preponderance of neutrophils; however, these cases may be complicated by the presence of a mixed bacterial infection. In places where these foci project against the capsule, there is thickening of the capsule as well as infiltration into the capsule by the inflammatory cells. Within these foci, tubules are destroyed with only remnants or occasional free epithelial cells remaining. Adjacent tubules frequently contain large numbers of these inflammatory cells. Cellular casts and leukocytes in the tubules were also reported by Jungherr (1944) and Mathews (1946). No thrombosis or infarction is observed.

The most striking change in the tubules is hydropic degeneration of the epithelium

FIG. 16.2 — Hydropic degeneration of renal epithelium which was apparent in all segments of the nephron unit except the glomerulus. Renal capsule at upper left corner. Hematoxylin-eosin. X 400.

(Fig. 16.2), which was also described by Bloom (1941) and Monlux (1948) in dogs infected with *L. canicola*. This is characterized by large, swollen, vacuolated cells in which the nuclei are crowded to the surface or are even invisible (fat stains on frozen sections using oil red O failed to reveal fat in the vacuoles). This degenerative change is present in all segments of the nephron unit except the glomerulus. In some tubules, epithelial cells are swollen to the extent that the lumen is closed. In others the cells are broken, permitting extrusion of the nucleus and cytoplasm into the lumen. Hyaline casts are not seen and tubules are not cystic. Rarely is mitosis of epithelial cells observed, and only an occasional tubule contains a double row of cells indicating hyperplasia. The relative lack of mitosis and the evidence of regeneration of epithelium were in contrast to lesions observed in dogs and cattle. The duration

FIG. 16.1 — Focus of neutrophils, monocytes, and lymphocytes at cortico-medullary junction. One tubule contains neutrophils. Many tubules (arrows) are undergoing degeneration. Hematoxylin-eosin. X 200.
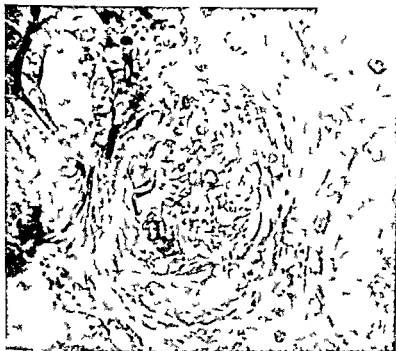
FIG 16 6 — Fibrosis of Bowmans capsule and capillary tuft Trichrome X 550

The only microscopic change observed in the liver was what appeared to be generalized cloudy swelling of liver cells In tissues from some animals hardly a cell could be found that was unaffected Langham et al (1958) referred to this foamy appearance of the liver cells and demonstrated by the Bauer Feulgen stain ing method that the change resulted from dissolution of glycogen Silver stains on liver sections failed to reveal any lepto spiras This was anticipated since lepto spiras tend to localize in the kidney tu bules after the appearance of the antibodies in the circulating blood

The gross and microscopic lesions are somewhat different in swine later in the course of the disease The following is a description of animals which had shed the organisms in the urine for six months and then became free of infection without treatment The antibody titer was grad ually decreasing at the time of slaughter No gross lesions were visible at the time of necropsy

Histopathological examination of the

FIG 16 7 — Proximal convoluted tubule filled with leptospiras which follow the contour of the free edge of the epithelium as well as forming a weblike mass across the lumen Silver stain X 1,100
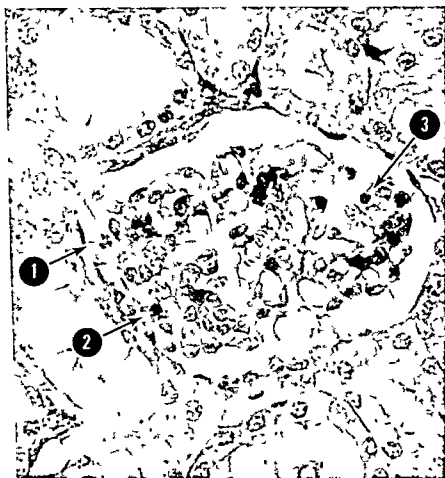
FIG 16 4 — Glomerulus show-
ing neutrophil (1), monocyte
(2), lymphocyte (3), and ad-
hesions at the left between
the tuft and Bowman's cap-
sule Note detritus in tubules
and Bowman's space Hema-
toxylin eosin X 575

masses in the lumens with no detail visible
They are located only in the lumen and
not intracellularly or interstitially The
masses of leptospiras usually follow the
contour of the free surfaces of the renal
epithelium which appears to give the cells
an irregular black border. However, they
are also found unattached in the lumen
or as weblike structures which extend com-
pletely across the lumen (Fig 16 7)

FIG 16 5 — Note dense, atrophic glomerular
tuft floating free in Bowman's capsule which al-
so shows swollen epithelium and some thicken-
ing of the capsule wall Trichrome. X 550

survival of the organism under natural conditions Thus preventing the spread of leptospirosis must include housing the animals in a sanitary environment which does not have puddles water holes or swampy areas

Ferguson and Powers (1956) and Ferguson et al (1956) demonstrated that swine can be kept in the same building and cared for by the same herdsmen without spreading leptospirosis from the infected to susceptible animals This depends on the simple precautions of preventing direct contact and drainage or splashing of urine from the infected to clean areas On the other hand natural infection in a herd of swine was transmitted from animals in one pen to susceptible swine in a second pen These pens were separated by a woven wire fence More than once the author saw a sow urinate in a position which deposited the urine in an adjoining pen

The spread of leptospirosis can be prevented by breaking the cycle of contact through infected urine between susceptible animals Consideration of this transmission cannot be confined to any one species on a farm where a mixed animal population is maintained Morter and Morse (1956) suggested that sheep may not be important in the transmission of leptospirosis but cattle swine and goats are involved

Because of the susceptibility of leptospiras to drying extremes of pH and other environmental factors leptospirosis should be one of the easiest diseases to control or eradicate In reality however this is extremely difficult because of the frequency of inapparent infection and the resulting unrecognized carriers This means that the use of laboratory facilities chiefly the serological test must be used to aid in the diagnosis Well defined segregation of infected carriers from susceptible animals must be carried out

This system of control based on the agglutination test and segregation can be supplemented by two agents which will aid in materially reducing the number of carriers These are (1) the use of a leptospiral bacterin and (2) antibiotic therapy

The first can be used to increase the resistance of swine likely to be exposed and the second procedure is of value in preventing or eliminating the kidney carrier stage

York and Baker (1953) reported the use of a killed suspension of *L pomona* cultivated in embryonated eggs for the prevention of leptospirosis in cattle Commercial bacterins or vaccines are now available to the veterinarian These products were developed for use in cattle but they have also been used in swine Ferguson et al (1956) used a leptospiral bacterin (Leptogen a product of Pitman Moore Co) in a large breeding herd of swine *L pomona* was introduced probably by an infected boar into a pen of 29 gilts The bacterin was used in 54 sows and boars and except for 3 sows in a pen adjoining the gilts which may have been infected at the time of vaccination there was no further spread of the disease

Although it is difficult to evaluate the field use of a product in a self limiting disease such as leptospirosis the author is aware of the use by veterinarians of the commercially prepared bacterins in swine The general impression is that gilts vaccinated at breeding time are protected from infection during pregnancy and therefore in a herd or community where *L pomona* is present they will be free of leptospiral abortions Certainly there is evidence that the bacterins induce a degree of immunity which will reduce the incidence of leptospirosis in an exposed herd

The induced resistance engendered by the leptospiral bacterin can aid in the ultimate control and eradication of leptospirosis caused by *L pomona* By reducing the number of susceptible swine the number of carriers will likewise be reduced

The leptospiras localized in the tubules of the kidneys in chronic carrier animals are not readily removed by chemotherapeutic agents In this location the organisms are protected from antibodies or other substances in the blood and tissues The only chemotherapeutic agent of value in these cases is one which is excreted by way of the proximal convoluted tubules where

kidney showed small foci of fibrosis as revealed by Masson's trichrome stain. In these fibrous areas tubules had disappeared, and increased numbers of capillaries were present, most of which contained blood.

Most tubules contained granular eosinophilic debris which may or may not be abnormal. In a few tubules small hyalinized casts were present but were not blocking the lumen. Albuminous degeneration was apparent in some tubules but this change was not general over the entire section. Tubules were not cystic and there was no evidence of infarction. Occasional small foci of lymphocytes were present in the interstitial tissue but monocytes and neutrophils were not seen.

Glomeruli had suffered the greatest permanent damage. Some capillary tufts remained only as small, dark, dense masses inside shrunken thickened Bowman's capsules. Other glomeruli had apparently retained their natural size but had undergone complete obsolescence with fibrosis which was continuous and indistinguishable from the thickened fibrous parietal layer of capsule. Many glomeruli were unaffected and remained functional, but even so in most of these there was some granular detritus in Bowman's space.

Silver stains did not reveal any leptospiras in these kidneys, and hamster inoculations including blind passages were not successful in isolating leptospiras. Bacteriological examination of these tissues did not reveal any other pathogenic agents.

Brief mention should be made of structures which might be confused with leptospiras in silver stained sections or in blood examinations. Fibrin strands faintly resemble leptospiras in silver stained sections except that the fibrin has a distinctly beaded appearance and may be present in long threads. It is also less dense than masses of leptospiras and usually it forms a loose, open network. Overtreatment of tissues with silver stains produces a dark background and leaves a precipitate which tends to be confusing.

CONTROL

The control and eradication of leptospirosis in swine is important not only because of the economic losses caused in this species of animals but also because swine are probably the most important reservoir of infection for other species. Except in the case of pregnant swine, leptospirosis exists very commonly in a herd without the owner's being aware of its presence. Not infrequently investigators (Bohl and Ferguson, 1952) have determined by serological test, the presence of leptospirosis in the swine on a farm only after clinical leptospirosis in the cattle had called attention to the problem.

The spread of leptospirosis depends upon the transmission of the leptospiras in the urine of an infected carrier to a susceptible host. The viable organism can enter the susceptible animals by any one of several routes. Experimentally the disease is readily produced by introducing the organisms into the conjunctival sac, the nasal cavity and the vagina (Ferguson and Powers 1956). Gillespie et al (1957) demonstrated that guinea pigs became infected when bathed with water contaminated with L. pomona. Ringen and Bracken (1956) produced infections in cattle by placing one of the feet, which had been shaved in a bucket of urine from a carrier cow. It seems logical to assume that under conditions commonly found in farm herds, viable leptospiras may enter the eyes nose, or abraded skin directly from the urine of an infected pen mate. Indirect infection may also result from contact with litter or water puddles contaminated by urine. Morter and Morse (1956) have demonstrated the ease with which pen contact with infected animals can spread the disease.

Gillespie et al (1957) demonstrated that L. pomona can survive for 10 days in surface water. They also demonstrated experimentally that creek water contaminated with positive bovine urine still contained active leptospiras after 15 days. The authors pointed out, however, that the surface waters in the Columbia Plateau area are alkaline, and this may influence the survival of the leptospiras. Even though waters in other parts of the country may not be so favorable for leptospirosis the presence of moisture is essential for the

as simple as indicated in the preceding paragraph There are differences in the virulence of strains of *L pomona* There are some 50 serotypes of Leptospira which are found in various parts of the world Only 8 of these serotypes have been defi nitely identified in the United States With world travel becoming increasingly com mon, both of man and animals vigilance

will be required to prevent the entrance or to limit the spread of other serotypes which may be much more virulent than *L pomona* Improvement of the methods for serological testing is urgently required to aid in identifying various strains of *L pomona* as well as any new species or sero types which may subsequently appear in this country

REFERENCES

BAKER C E GALLIAN M J PRICE K E AND WHITE E A   1957 Leptospirosis I Therapeutic studies on the eradication of renal carriers of porcine leptospirosis Vet Med 52 103

BLOOM, F 1941 The histopathology of canine leptospirosis Cornell Vet. 31 266

BOHL E H AND FERGUSON L C 1952 Leptospirosis in domestic animals Jour Amer Vet Med Assn 121 421

————, POWERS T E AND FERGUSON L C 1951 Abortion in swine associated with lepto spirosis Jour Amer Vet Med Assn 121 262

BRUNNER K T AND MEYER K F 1949 Streptomycin in the treatment of leptospiral carriers Proc. Soc Exper Biol and Med 70 450

BRYAN, H S 1957a Studies on leptospirosis in domestic animals VI Vaccination of swine with *Leptospira pomona* bacterin Vet Med 52 51

———— 1957b Studies on leptospirosis in domestic animals VII A rapid plate agglutination test for *Leptospira pomona* Vet Med 52 111

———— RHOADES H E AND WILLICAN D A 1953 Isolation of *Leptospira pomona* from aborted swine fetuses Vet Med 48 438

CHANG S L 1947 Studies on *Leptospira icterohemorrhagiae* I Two new mediums for growing *L icterohemorrhagiae L canicola*, and *L biflexor* and a method for maintaining the virulence of *L icterohemorrhagiae* in culture Jour Infect Dis 81 28

CLAYTON G E B DERRICK E H AND CILIENTO R W 1937 The presence of leptospirosis of a mild type (seven day fever) in Queensland Med Jour Australia 1 647

FERGUSON, L. C BOHL E H AND POWERS, T E 1953 Leptospirosis in swine Proc. U S Live stock San Assn , p 332

———— LOCOCO, S SMITH, H R AND HAMDY, A. H 1956 The control and treatment of swine leptospirosis during a naturally occuring outbreak Jour Amer Vet. Med Assn 129 263

————, AND LOWERS T E 1956 Experimental leptospirosis in pregnant swine Amer Jour Vet Res 17 471

FIELD, H I , AND SELLERS, K C 1951 *Leptospira icterohaemorrhagiae* infection in piglets Vet Rec. 63 78

GILLESPIE R W H, KENZY, S G RINGEN L M and BRACKEN F K 1957 Studies on bovine leptospirosis III Isolation of *Leptospira pomona* from surface waters Amer Jour Vet Res 18 76

GOCHENOUR, W S JR., JOHNSTON, R V YAGER R H AND GOCHENOUR W S 1952 Porcine leptospirosis Amer Jour Vet Res 13 158

GSELL, O 1946 Leptospirosis pomona die Schweinehuterkrankheit Schweiz med Wschr 76 237

HADLOW, W J AND STOENNER, H G 1955 Histopathological findings in cows naturally infected with *Leptospira pomona* Amer Jour Vet Res 16 45

JUNGHERR, E 1944 Bovine leptospirosis. Jour Amer Vet Med Assn 104 276

KELSER R A AND SCHOENING H W 1948 Manual of Veterinary Bacteriology 5th ed Williams and Wilkins Co Baltimore.

KLARENBEEK A , AND WINSSER J 1937 Ein Fall von spontaner Weilscher Krankheit bei Ferkeln Deutsch tierarztl Wschr 45 431

KRELIKOGORSKAIA T A., AND REMENTSORA M M 1957 The isolation of strains of leptospires from the tick *Dermacentor marginatus* from cattle. Jour Micro Epid and Immunobio 28 251 (Abstr in Vet Med 52 545)

LANGHAM R F MORSE E V., AND MORTER R L 1958 Experimental leptospirosis A Pathology of *Leptospira pomona* infection in swine Amer Jour Vet Res. 19 355

LILLIE, R D 1954 Histopathologic Technic and Practical Histochemistry Blakiston Co., New York

McMANUS, J F A 1950 Medical Diseases of the Kidney Lea and Febiger Philadelphia

MATHEWS F P 1946 A contagious disease of cattle associated with leptospira Amer Jour Vet Res 7 78

the leptospiras are located (Weber et al 1956) Penicillin is of no value in the chronic carrier animal (Brunner and Meyer, 1949) however, streptomycin and the tetracycline group of antibiotics are effective

Weber et al (1956) using hamsters which had a chronic kidney infection caused by L. canicola demonstrated that dihydrostreptomycin at levels of 15 mg per kg of body weight per day for 3 days eliminated the organisms from the urine These workers also cleared the kidneys with somewhat higher levels of oxytetracycline tetracycline and erythromycin

Sidler (1954) reported that in controlled experiments pigs weighing about 100 lb were cleared of kidney infection with as little as 0 25 gm of streptomycin Studies in the United States (Ferguson et al 1955) indicate that a single intramuscular injection of streptomycin at a level of 10 mg per kg body weight will eliminate the leptospiras from the kidneys in most cases Ferguson et al (1956) reported that chlortetracycline fed at a level of 100 gm per ton of feed or about 1 gm per sow per day for 10 days eradicated the kidney carrier stage in only about one half of the treated group Baker et al (1957) reported that oxytetracycline given at the rate of 500 to 1 000 gm per ton of feed for 7 days eliminated the renal carrier stage in 6 of 7 pigs The authors suggested that 94 per cent of animals treated at these levels would be expected to eliminate the leptospiras

By means of these methods of control aimed at eliminating the carrier animals losses from leptospirosis can be greatly reduced Particular emphasis must be placed on the danger of introducing a boar into a susceptible herd of swine without first testing the animal for leptospiral antibodies The same should apply to any additions but the common practice of adding a boar from another herd at the beginning of the breeding season increases the probability of spreading leptospirosis

Although there is little information available on the duration of immunity in swine following vaccination many veterinarians are recommending that sows be revaccinated at each breeding period (York, 1957) This is especially important in communities where the infection is known to be prevalent or where there is a movement of animals from place to place

The ease of cross infection between species of animals and the common occurrence of localization in the kidneys with no clinical manifestations add to the difficulties encountered in controlling leptospirosis There may be little gained if one concentrates only on the swine on an infected farm when there may be carrier cattle sheep goats and horses available to maintain a source of infection Moreover, one should not overlook the possible role of wild animals such as deer and various rodents as carriers of L pomona A recent report (Kreplogorskaia and Rementsorn 1957) indicates still another possible method of spread two strains of L grippotyphosa were isolated from the tick Dermacentor marginatus

CONCLUSION

L pomona has been found in at least 11 of the states (Steele et al, 1957) Obviously, the disease has been widely disseminated even though it was first recognized as an important disease in swine only since 1950 It can be controlled by rigid sanitary procedures involving all species of animals on the premises The serological test is very useful in determining the probable presence of the pathogen and thus serves as an aid in segregating the infected and susceptible animals The use of a leptospiral bacterin is of value in preventing losses from abortion in pregnant swine and it will also reduce the number of potential carriers Antibiotic therapy may be used in selected herds to eliminate the leptospiras from the kidneys of infected carriers The combination of these methods, if wisely and persistently used can serve in the complete eradication of L pomona infection

Eradication of leptospirosis may not be

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CHAPTER 17

Brucellosis

Brucellosis of swine, formerly called contagious abortion of swine, is an infectious disease that has been recognized as a specific entity since 1914 when Traum (1914) isolated *Brucella suis* from aborted fetuses. Results of studies that have occurred from time to time on the incidence of brucellosis of swine show a difference which appears to be related to the location where the swine sera were obtained. Although the incidence of the disease in the United States is not definitely known, a recent and more comprehensive report (U S D A, 1957) than those in the past show the average infection to be 5.2 per cent. Available evidence indicates that brucellosis occurs in most swine raising areas of this country and in most countries throughout the world where swine exist either in the wild or in the domestic state.

The principal natural host for *Br suis*, the leading cause of swine brucellosis, is the pig. Although *Br suis* infection occurs naturally in horses, cattle, dogs, and fowl, the disease is much more self limiting in these animals than in swine. This is doubtlessly associated with natural resistance of foreign hosts to *Br suis*. Most studies on the susceptibility of dogs and chickens to brucellosis have been concerned with the species *Br abortus*, however, it has been demonstrated that both species of animals are susceptible to *Br suis* and *Br meliten*

sis More recently, the European hare has been incriminated as a natural host for *Br suis* (Christiansen and Thomsen 1936. Fritzsche, 1956). Danish investigators (Bendtsen *et al*, 1934, 1956) believe that this animal is a potential reservoir of infection and is responsible for outbreaks of brucellosis of swine in Denmark.

The guinea pig is the most susceptible of the experimental animals to the three species of Brucella. Although domestic rabbits, hamsters, and white mice are susceptible to these infections, the ensuing disease is more self limiting in them than in guinea pigs.

Very little is known about the susceptibility of goats and sheep to *Br suis* because the opportunity for exposure is exceedingly slight under usual management practices. White and wild rats are relatively insusceptible to *Br suis*.

ETIOLOGY

Brucella suis is recognized as the principal cause of brucellosis of swine because it is the species of Brucella most frequently isolated from swine in herds naturally affected with brucellosis. *Brucella melitensis* and *Brucella abortus*, however, also are capable of causing infection in swine under natural conditions of exposure. Isolation of *Br melitensis* from naturally infected swine in the United States was first

[273]

Mochtar, A   1940   Over het voorkomen von leptospiras bij varkens te Batavia   Geneesk tijdschr v Nederl Indie. 80 2334

Monlux W S   1948   Leptospirosis IV The pathology of canine leptospirosis   Cornell Vet 38 199

Morse, E V Allen, V Krohn A F and Hall R   1955   Leptospirosis in Wisconsin I Epizootiology and clinical features   Jour Amer Vet Med Assn 127 417

——   Bauer D C Lancham R F Lanc R W and Ullrey, D E   1958   Experimental leptospirosis IV Pathogenesis of porcine *Leptospira pomona* infections   Amer Jour Vet Res 19 388

Morter R L. and Morse E V   1956   Experimental leptospirosis II The role of calves in the transmission of *Leptospira pomona* among cattle swine sheep and goats   Jour Amer Vet Med Assn 128 408

Nisbett D 1   1951   *Leptospira icterohemorrhagiae* infection in pigs   Jour Comp Path and Therap 61 155

Reinhard K R   1951   A clinical pathological study of experimental leptospirosis in calves   Amer Jour Vet Res 12 282

Ringen, L. M and Bracken F K   1956   Studies on bovine leptospirosis II The effect of various levels of tetracycline hydrochloride on bovine leptospirosis   Jour Amer Vet Med Assn 129 266

Runnells R A   1954   Animal Pathology 5th ed Iowa State College Press Ames Iowa

Ryley J W and Simmons G C   1954   *Leptospira pomona* as a cause of abortion and neonatal mortality in swine   Queensland Jour Agr Sci 11 61

Sanger V L   1957   Personal communication

Savino E and Rennella E   1945/48   Leptospira en cerdos de la República Argentina   Rev d Inst Bact 13 66

Schirren C G   1953   Experimentelle Erzeugung von Pseudospirochaeten in Blut   Dermatolog ca 107 238

Sidler W   1951   Epidemiologische und therapeutische Untersuchungen uber die Leptospire der Schweine   Inaugural Dissertation Univ of Bern Bern Switzerland

Steele J H Galton M M and Menges R W   1957   Leptospirosis as a world problem   Vet Med 52 517

Stoenner H G   1951   Application of the capillary tube test and a newly developed plate test to the serodiagnosis of bovine leptospirosis   Amer Jour Vet Res 15 434

——   1955   Application of serology to the diagnosis of leptospirosis   Proc. Book Amer Vet Med Assn p 172

Weber W J. Creamer H R and Bohl E. H   1956   Chemotherapy in hamsters chronically infected with *Leptospira canicola*   Jour Amer Vet Med Assn 129 271

York C. J   1957   Immunology and prophylaxis of leptospirosis   Vet Med 52 563

—— and Baker J A   1953   Vaccination for bovine leptospirosis   Amer Jour Vet Res 14 5

W H O , 1953) Each of these characteristics is determined by employing specific tests It is emphasized that no single test is entirely dependable consequently, classification of a Brucella culture is based on the combined findings obtained with all the tests

Neither *Br suis* nor *Br melitensis* require increased CO_2 tension for growth whereas original isolations of *Br abortus* require increased CO_2 tension approximately 10 per cent above that of atmospheric air

All strains of *Br suis*, except those of the Danish variety, produce large amounts of H_2S for 4 or 5 days, *Br melitensis* produces very little or no H_2S, and *Br abortus* produces moderate amounts for 2 days or longer

Growth of *Br suis* is inhibited by basic fuchsin and not by thionin growth of *Br melitensis* is not inhibited by either dye and growth of *Br abortus* is inhibited by thionin and not by basic fuchsin (Huddleson, 1943) Occasionally strains of each species do not conform to the regular pattern The concentration of each dye may vary with the kind of media employed Numerous studies have been made on the dye sensitivity test for differentiating the species of Brucella since it was first introduced by Huddleson (1929)

In general, *Br suis* shows a positive reaction for urease activity (Hoyer, 1950) immediately or within 15 to 30 minutes after incubation and most *Br abortus* strains require 2 hours or longer The test has limited value in identifying *Br melitensis* because the urease activity of some strains approaches that of *Br abortus* The strains of *Br melitensis* isolated from swine in the midwestern states show greater urease activity than the strains isolated from goats

Catalase activity (Huddleson, 1943) is greatest in *Br suis* and least in *Br abortus*, with that of *Br melitensis* being somewhere between the other two species

Sodium diethyldithiocarbamate-impregnated (Renoux, 1952a) filter paper discs placed on solid agar medium completely inhibit the growth of *Br suis* adjacent to the disc, thus producing a clear zone without a peripheral ring It inhibits the growth of *Br melitensis* only slightly adjacent to the disc, but forms a white ring at the periphery of this area and completely inhibits growth in a zone outside of the ring The growth of *Br abortus* is not inhibited next to the disc, but a brown and a white ring are formed at the periphery of this area, with complete inhibition of growth in a zone outside the white ring

Brucella suis and *Br abortus* cannot be distinguished from one another with monospecific sera, but they can be differentiated from *Br melitensis* The success of this test is dependent on production of antiserum with smooth cultures of *Br abortus* or *Br melitensis* followed by the proper degree of absorption of antisera with the smooth heterologous organisms (Evans, 1923, Huddleson, 1943)

CLINICAL SIGNS

Most investigators generally agree on the symptoms associated with brucellosis of swine Any disagreements that exist are doubtlessly related to differences in experimental methods, in stage of the disease, and in herd management.

Repeated and prolonged studies by the writer and co workers (Manthei 1957) failed to show any consistent rise or undulating type of temperature during the course of *Br suis* infection in swine

Bacteremia was one of the first signs of infection following exposure (Cotton and Buck 1932, Hutchings, 1950a) and is most persistent the first 8 weeks following exposure Not all swine that show a bacteremia develop clinical manifestations of brucellosis or localization of infection Either *Br suis* infection fails to establish itself in approximately 15 per cent of swine that show a bacteremia or the swine recover from the disease within a short time Since this condition occurs in non vaccinated as well as in vaccinated swine, it would appear that both natural and acquired immunity are related to the phenomenon Huddleson (1955), writing on the subject of bio-

reported by Borts et al in 1946 There was some evidence presented by Jordan and Borts (1946) and Huddleson (1943), however that *Br melitensis* existed in swine prior to this date Huddleson classified 2 of 132 strains of *Brucella* isolated from swine as *Br melitensis* prior to 1943 Since that time several investigators have isolated this species of *Brucella* from swine McCullough et al (1949, 1951) were the first and only investigators to report isolation of *Br abortus* from naturally infected swine Originally, it was believed that *Br abortus* was responsible for brucellosis of swine (Good and Smith 1916) Although *Br abortus* infection has been produced in swine experimentally, the results have been variable regardless of the method of exposure employed

As all three species of *Brucella* are involved in the cause of swine brucellosis, the discussion of their characteristics will be one mainly of comparison (Huddleson 1943 Merchant and Packer, 1956) The cellular and colonial morphology of the three species are similar in most respects The size and shape of cells may vary slightly between strains of the same species, as well as between each species *Brucella suis* organisms are bacilli that vary in size from 0 6 to 3μ in length and 0 4 to 0 8μ in width *Brucella melitensis* organisms usually occur as coccoid, or short bacillary forms They vary in length from 0 4 to 2 2μ and in width from 0 4 to 0 8μ *Brucella abortus* usually occur as short bacilli, but may occur as coccoid forms The length varies from 0 4 to 2 5μ and the width from 0 4 to 0 6μ *Brucella* organisms are non motile and do not form endospores They are stained with the aniline dyes and are Gram negative Although there is some disagreement concerning the presence of a capsule on *Brucella*, capsules are readily demonstrated on the cells of smooth and intermediate colonies of the three species by the india ink staining technique described by Huddleson (1941b)

Original isolations of the three species of *Brucella* appear as small, convex, and translucent colonies on the surface of agar media, and they are semitransparent by obliquely transmitted light All smooth forms of *Brucella* dissociate into intermediate, rough, or mucoid forms under certain artificially induced environmental conditions

Most *Br suis* organisms grow more rapidly and luxuriantly than either *Br melitensis* or *Br abortus* organisms on the various kinds of artificial media Colonies of *Br suis* are usually distinguishable on the surface of suitable media after three days of incubation at 37° C, whereas those of the other two species are not visible until about the fourth to the seventh day *Br suis* and *Br melitensis* are aerobes, and *Br abortus* is a facultative anaerobe Only *Br abortus* requires an increased CO_2 tension of approximately 10 per cent by volume above that of atmospheric air for primary isolation from tissues, excretions and secretions of animals (Ardrey, 1941, Huddleson, 1943)

The optimum pH for growth of *Brucella* varies from 6 6 to 7 4, depending on factors such as the buffering system, type of medium, rate of growth, and time of observation

Most *Brucella* have specific nutritional requirements for optimum growth These requirements have been reported in detail by other investigators and consequently will not be discussed in this chapter except to state that a suitable medium must contain an adequate source of nitrogen, carbon and energy Because most diagnostic laboratories are not equipped to prepare special media, there are several commercially prepared media available that are satisfactory for propagation of *Brucella* These are Tryptose, Trypticase soy, and Albimi There is additional information available on the physiology and chemistry of *Brucella* that will not be discussed in this chapter (Cameron et al, 1952, Gee and Gerhardt, 1946, Glassman and Elberg 1946, Hoyer, 1950, Pennell, 1950)

One of the most important parts of a discussion on etiology of brucellosis is the proper identification of species of *Brucella* by their biochemical and serological characteristics (Huddleson, 1943, Hulse, 1952 Renoux, 1952b, Rep 2nd Session F A O /

months of sexual rest, the conception rate is usually good Sows with a persistent genital infection, however, seldom conceive Infertility and lack of sexual drive in boars are most frequently associated with infection of the testicles Boars that have little or no testicular involvement but have infection of the accessory genital organs may disseminate large numbers of *Br suis* in the semen yet are not necessarily sterile According to Andrews and Hutchings (1946), the quality of semen from infected boars is poor It has been the experience of the writer that boars with localized infection in the seminal vesicles are the most prolific disseminators of *Br suis* in the semen Frequently, boars of this kind are considered infertile because of the low conception rate in susceptible sows that they have bred, but infertility is actually associated with genital infection in the sows

Clinical evidence of brucellosis in suckling and weanling pigs is limited to relatively low agglutinin titers and temporary bacteremia Swollen joints and lameness occasionally are observed Orchitis seldom develops before boar pigs approach sexual maturity, which is about five or six months of age Abortions are exceptionally rare in sows infected as pigs (Goode et al, 1952, Hutchings et al, 1946a, Manthei et al, 1952, Thomsen, 1934)

Although *Br melitensis* has been incriminated as a naturally occurring cause of brucellosis in swine in the United States knowledge of the natural course of this infection is meager when compared with that of *Br suis* Hutchings et al (1951) reported typical symptoms of brucellosis in a herd of swine infected with an organism identified as *Br melitensis* Artificial exposure of swine with a strain of *Br melitensis* originally isolated from swine by Hoerlein (1952) provides us with additional information on the pathogenesis of this type of infection When these results are compared with those of the writer (Manthei, 1957), who used *Br suis* as the exposure material under comparable conditions, it would appear that *Br suis* is more pathogenic for swine than *Br meli-*

tensis Since these data have not been published, a summary of the results follows intravenous exposure—*Br suis* was isolated from 6 of 8 pregnant sows, and 5 aborted conjunctival exposure—*Br suis* was isolated from 19 of 27, and 10 aborted, and intravaginal exposure—*Br suis* was isolated from 10 of 15, and 11 aborted In addition, the Brucella agglutinin titers appeared more rapidly and developed to a higher level in these animals than in those exposed to *Br melitensis* by Hoerlein (1952) Hoerlein also reported that udder infection could not be demonstrated in the exposed sows after farrowing and that routine vaginal swabs were negative for Brucella in all cases Moreover, the incidence of localized infection was relatively low at the time of necropsy and culture preparation, which was 6 to 12 months following exposure When these results are compared with those from studies of *Br suis* infection by Hutchings (1950a) and Manthei (1948), *Br melitensis* appears to be the least pathogenic for swine of these two species of Brucella, and produces a more self limiting infection

Our knowledge of the pathogenesis of *Br abortus* in swine is very limited More over, the results of artificially inducing this type of infection in swine are sufficiently different from those obtained from infections induced with *Br suis* and *Br melitensis* to draw any definite conclusions Although McCullough et al (1949, 1951) established the presence of naturally occurring *Br abortus* infection in swine, it has been difficult to reproduce the disease experimentally Graham et al (1930) failed to produce *Br abortus* infection in gilts by feeding them milk from infected cows, and Gilman et al (1934) were unable to produce infection in swine by feeding a suspension of *Br abortus* but were successful in producing infection by intravenous injection Subsequently, Washko et al (1951) and Bay et al (1951) were successful in experimentally producing Br abortus infection in swine, but interpretation of results is complicated by isolation of *Br suis* from some of the animals Goode and Manthei (1953) ex

chemical and histopathological reactions in the evolution of bovine brucellosis, states that our knowledge is incomplete concerning the fate of pathogenic Brucella after they penetrate the body and enter the blood stream but do not produce an apparent disease Bacteremia is seldom demonstrated longer than 21 days or more than two intermittent times in animals that do not show symptoms whereas it usually persists much longer in swine that develop apparent brucellosis Intermittent bacteremia has been demonstrated from 1 to 34 months, with the average being 8 months, in sows showing clinical manifestations of the disease

The development, concentration, or persistence of antibodies, particularly agglutinins, in the blood serum following exposure to virulent Br suis varies considerably between individuals These variations are related to method and amount of exposure susceptibility of the animals, and site of localization of infection A diagnostic level of agglutinins usually does not develop prior to 10 days, and maximum agglutinin titers seldom develop prior to 21 days in swine following exposure to Br suis Agglutinin titers usually develop most rapidly in swine exposed intravenously and intracutaneously, most slowly in those exposed per vagina by either natural service or artificial insemination, and at some time between the two extremes in those exposed per os or per conjunctiva Maximum agglutinin titers are generally higher and persist longer in adults than in suckling or weaning pigs Most swine show Brucella agglutinins in the 1 100 or higher dilution of serum at some time following infection, however, there is a tendency for titers to decline or become transient as animals are in the process of recovering from the disease or as the disease becomes chronic (Creech, 1930, Hutchings, 1950b)

Clinical evidence of Br suis infection may vary considerably in different herds These variations are influenced by factors such as general susceptibility of swine, stage and number of pregnancies virulence of the infectious agent, method of exposure, and site of localization of infection The classical clinical manifestations of Br suis infection are abortion, birth of stillborn or weak pigs, infertility, unilateral or bilateral orchitis, posterior paralysis, and lameness Decreased sexual drive is occasionally observed in affected boars

Abortions have been observed as early as 22 days following natural service to boars disseminating Br suis in the semen Early abortions are usually overlooked under field conditions, and the first indication of infection is a large percentage of sows or gilts showing signs of estrus 30 to 45 days after the service that terminated in conception Little or no vaginal discharge is observed with early abortions Abortions that occur during the middle or late stages of gestation are usually associated with females that acquire infection after pregnancy has advanced past 35 or 40 days The persistence of genital infection in females varies considerably Brucella suis usually persists a minimum of one month in the nongravid uterus In a group of sows that were bred to boars disseminating Br suis in the semen, several have shed Br suis in vaginal discharge for at least 30 months An apparent abnormal vaginal discharge is seldom observed in sows that have uterine infection The percentage of females that eventually recover from genital infection is relatively high

Genital infection is much more persistent in boars than in sows The writer (Manthei, 1957) has studied 6 boars, 5 of which have shown persistent genital infection for 3 years and one for more than 4 years All of the evidence strongly indicates that boars infrequently or never recover from genital infection

The length of time that boars and sows remain infertile is directly related to the duration of genital infection and the extent of pathological changes (Connoway et al, 1921, Crawford and Manthei, 1948 Hutchings and Andrews, 1946 Thomsen, 1934) When genital infection does not persist longer than one month following abortion normal parturition, or breeding, and sows are permitted several

FIG 17 1 — The top specimen is the testicle of a boar showing multiple abscesses of the epididymus The lower testicle is normal

FIG 17 3 — Cystic uterus associated with Brucella suis infection in a sow

FIG 17 2 — Seminal vesicle infection in the boar Only one lobe is infected The prostate and testicles are normal

FIG 17 4 — The uterus shown in Figure 17 3 opened to show the cystic condition of the uterine mucosa

posed two groups of sexually mature gilts to virulent *Br abortus* for a period of 60 to 90 days Only 1 of 5 gilts exposed through contact with infected cattle and 1 of 10 fed milk of cows that contained *Br abortus* developed diagnostic agglutinin titers *Brucella abortus* was isolated from the retropharyngeal lymph glands of one gilt from the latter group The limited evidence available suggests that *Br abortus* is not highly pathogenic for swine, and swine are not likely to show clinical evidence of the disease when infection becomes established

PATHOLOGY

Macroscopic pathological changes produced by *Br suis* in swine are quite variable Abscess formation is common in affected organs and tissues In the case of orchitis, single or multiple abscesses are frequently observed in the parenchyma and in the epididymis (Fig 17 1) Enlargement and abscess formation are usually associated with localization of infection in *the seminal vesicles (Fig 17 2)* Although localized *Br suis* infection has been demonstrated in the bulbo urethral gland, macroscopic pathology is usually absent Thomsen (1934) reported miliary abscesses of the mucosa of the uteri of sows with genital infection We have observed only one case of miliary brucellosis of the uterus, but we have observed multiple cysts in the mucosa of a number of uteri from which *Br suis* was isolated (Figs 17 3 and 17 4) *Brucella suis* has likewise been isolated from ovaries showing multiple cysts Catarrhal inflammation of the uterine mucosa is more frequently observed than is pyometra

Abscesses or necrosis of the intervertebral disc and adjacent bone structure of the vertebrae are associated with spondylitis Locomotion may or may not be impaired, depending on the degree of involvement of the spinal cord A description of the vertebral lesions of swine caused by *Br suis* was first reported by Creech (1930) and, subsequently, by Feldman and Olson (1933)

Although *Br suis* produces macroscopic

lesions of parenchymatous organs, it is frequently isolated from these organs when gross pathology is absent Lesions usually appear as encapsulated nodules, varying in size and number, containing homogenous yellow or whitish gray pus, or caseous material Anderson and Davis (1957) have studied a number of cases of nodular splenitis of swine and suggest that this condition in the absence of other lesions justifies a presumptive diagnosis of brucellosis Although the writer (Manthei, 1957) has observed nodular splenitis of swine that was associated with *Br suis* infection, the frequency of its occurrence was too low to be highly significant in diagnosing brucellosis of swine Furthermore bacteriological examination of various tissues obtained from 75 infected swine at the time of necropsy, *Br suis* was isolated from the spleen of 30 per cent, only a few of which showed nodular lesions Other organs that occasionally show nodular lesions are liver, kidneys, and lymph glands *Brucella suis* has been isolated from abscesses found in the thoracic, abdominal, and pelvic cavities, as well as from various external areas of the body Catarrhal inflammation or abscesses of the tendon sheaths or joint capsules are occasionally observed (James and Graham, 1930)

Recorded observations of the macroscopic pathology of *Br melitensis* and *Br abortus* infection in swine are practically nonexistent The only suggestive evidence of differences in gross lesions produced by each of the three species of *Brucella* is in guinea pigs Nodules and abscesses are observed more frequently with *Br suis* than with *Br melitensis* and *Br abortus* infections Regardless of this evidence it would appear unwise to speculate on the type of lesions that may be produced in swine by *Br melitensis* or *Br abortus* until a thorough study is made of the two infections in this animal

Development of knowledge of the microscopic pathology of *Br suis* infection in swine has been related principally to specific macroscopic pathological conditions and not to the sequence of events in the evolution of histopathological changes

FIG. 17.6 — Higher magnification of a section from Fig. 17.5. X 110. (W. A. Anderson and C. L. Davis, J. A. V. M. A., 1957.)

undergo some calcification, and abscesses may become encapsulated with fibrous tissue. Brucella organisms may remain within the lesions for long periods of time. Based on the microscopic lesions reported by Creech (1930, 1935, 1936), Feldman and Olson (1933), and Anderson and Davis (1957), it does not seem out of order to assume that the evolution of histopathology produced by *Br. suis* in swine may be similar to that observed in small experimental animals. If the same situation should be true regarding *Br. abortus*, it would explain the benign character of this infection in swine. Furthermore, since some strains of *Br. melitensis* produce lesions similar to that produced by *Br. suis* and other strains act more like *Br. abortus*, it is possible that clinical manifestations of *Br. melitensis* infection of swine occur less frequently or are less obvious than those of *Br. suis*. This

information may help to explain why the majority of serious outbreaks of brucellosis of swine are caused by *Br. suis*.

DIAGNOSIS

The most accurate method of diagnosing brucellosis of swine is isolation and classification of the species of Brucella involved. This is accomplished by direct culture of specimens on a suitable medium or inoculation of specimens into guinea pigs and subsequent direct culture of their tissues and seroagglutination test of their blood sera. Many times, however, this is not feasible because laboratory facilities are inadequate or unavailable, or it is impossible to obtain specimens for examination. Moreover, the time required to conduct the necessary studies generally makes the procedure impractical in a large scale control program.

in tissues in the natural host. This is not to infer that our knowledge of the histopathology of brucellosis is totally lacking, because a number of investigators have made thorough studies of brucellosis in man, dogs, guinea pigs, mice, rabbits, and chick embryos. An excellent review of histological reactions in the evolution of bovine brucellosis has been published by Huddleson (1955).

As early as 1912, a very thorough and accurate report of the basic microscopic lesions of brucellosis in guinea pigs was made by Fabyan (1912) and few original observations have been added since that time. In general, there is agreement concerning granulomas in tissues containing large numbers of reticuloendothelial cells, occurrence of occasional giant cells, and presence of intracellular Brucella.

More recently, Braude and Anderson (1950) and Braude (1951) reported on the sequence of events observed in the origin and development of hepatic granulomas due to *Br. abortus*. Within a few hours after *Brucella* was introduced into the animals, organisms were observed in the cytoplasm of polymorphonuclear leukocytes and epithelioid cells parasitized with *Brucella* in the sinusoids of the liver. These

events were followed by focal aggregations of parasitized epithelioid cells and a marked reduction of polymorphonuclear leukocytes. Cellular aggregates increased in size within the sinusoids to form early granulomas, and intracellular organisms disappeared. Granulomas fused to form large focal lesions. Three months later, the center of the granulomas showed hyaline changes and the appearance of some giant cells. These changes were followed by a gradual decrease in the size of the granulomas and in the surrounding cellular inflammation. Finally, no evidence of histopathological changes could be seen in tissues examined one year after the animals were exposed to *Br. abortus*.

The sequence of initial events were similar for *Br. suis*, but marked differences were noted in subsequent changes. Arrival of parasitized polymorphonuclear leukocytes in hepatic sinusoids was not followed by appearance of epithelioid cells or reduction in the number of *Brucella*, but by an accumulation of polymorphonuclears and destruction of tissue resulting in the formation of caseous centers that suppurated. There was a sharp line of demarcation between areas of necrosis and surrounding tissue. Caseous centers may

FIG. 17.5 — A subcapsulated lesion protruding from the surface of a swine spleen showing the caseous center, calcium deposits, cellular infiltration, and capsule. X 30. (W. A. Anderson, and C. L. Davis, J. A. V. M. A, 1957.)

to non infected herds, titers of 1 25 and 1 50 also were observed in animals that had not been exposed to Brucella Consequently, a number of workers have recommended that interpretation of the seroagglutination test should be based on agglutinin titer profile and history of a herd The present accepted interpretation of the test is that animals with no titers as well as those with titers of 1 25 and 1 50 are potentially infected if they are associated with other swine, some of which show titers of 1 100 or higher Moreover a herd that does not contain any animals with titers above 1 25 or 1 50 on repeated tests and does not show any other evidence of brucellosis, should be considered free of the disease Therefore, the foregoing information supports the contention that the seroagglutination test is more useful in diagnosing infection in the herd than in the individual

Investigators have isolated and identified the nonspecific substance in sera of cattle or developed tests which appear to differentiate between nonspecific and brucellar agglutinins Hoerlein (1953b) has suggested a modification of the standard tube agglutination test by incubating the serum and antigen mixture in standard dilutions at 56° C for 16 hours Serum samples from herds known to be free of brucellosis but reacting to the standard tube agglutination test receded to negative or to a much lower titer when tested by the modified method whereas serum samples from swine infected with *Br suis* or *Br melitensis* did not show a marked decrease in titer Regardless of these encouraging results Hoerlein has suggested that more exhaustive studies should be made of the modified test before it is recommended as a standard diagnostic procedure

A new procedure, recently introduced by Rose and Roepke (1957) for the detection of nonspecific agglutinins in cattle sera, appears to be worthy of consideration for the same purpose in swine sera This procedure involves the use of acidified antigens of different hydrogen ion concentrations in conjunction with the plate agglutination technique As the hydrogen

ion concentration of the Brucella antigen is increased, the greater is the tendency for agglutinin titers to decrease

Another test that has shown promise in differentiating between nonspecific and brucellar agglutinins in the blood sera of cattle is a modification of the tube agglutination by investigators at Beltsville The basis for modification is similar in some respects to that proposed by Hoerlein (1953b) however, there are marked differences in incubation temperature incubation time, and final preparation of serum antigen mixture for reading and interpretation The amount of research on sera of swine is too limited to do other than suggest its possible application

Allergic tests have been considered and studied but the results have not stimulated much enthusiasm especially from the standpoint of replacing the seroagglutination tests Three different allergens prepared and supplied by Huddelson have been tested on infected and noninfected swine at the Animal Disease Station Beltsville, Maryland These were a phosphatide fraction of Brucella cells, a purified culture filtrate of *Br suis* and a soluble nucleoprotein fraction In general the results show that all of the allergens were slightly more sensitive than the seroagglutination tube and plate tests however both diagnostic methods failed to identify a small percentage of infected animals and erroneously classified some noninfected animals Very little difference was noted in the intensity of reactions in naturally infected and in artificially infected swine It should be emphasized however, that the naturally infected swine were from a herd that had experienced a relatively recent outbreak of brucellosis This is mentioned because Delez *et al* (1947) reported that allergic reactions were greatest in artificially sensitized swine They also point out that more reactions were obtained with the allergic test than with the seroagglutination test in a herd of swine that had been sensitized recently by natural exposure, whereas the reverse was true in another herd of older swine that had been sensitized for a longer period of time

The seroagglutination test is the most practical method of diagnosing brucellosis of swine at the present time Many re search workers have contributed to the development of the agglutination test to the degree where it is one of the most re liable diagnostic tools in use today De velopment of a practical method of col lecting blood from swine by Carle and Dewhirst (1942) has increased the practi cability of the seroagglutination test Hoer lein et al (1951) published additional in formation that is helpful on procurement and handling of swine blood samples Both tube and plate methods are equally ef ficient however, hemolysis of blood serum causes less interference with interpreta tion of the plate than of the tube test The same standard *Br abortus* tube and plate agglutination antigens produced by Agricultural Research Service and em ployed in the United States for the diag nosis of bovine brucellosis are used for the diagnosis of swine brucellosis Comparisons of standard plate and tube antigens pre pared from *Br abortus* strain 1119 were made with antigens prepared from the same strain of *Br suis* that was used to expose swine experimentally Comparative tests on more than 2,000 swine serum samples definitely showed that antigens prepared from *Br abortus* were equal in all respects as diagnostic agents to those prepared from the homologous strain of *Br suis* (Manthei, 1957)

Although the seroagglutination test has some limitations in diagnosing brucellosis of individual swine, actually it may be more correct to associate these limitations with the individual animal than with the test Generally speaking, agglutinins do not reach a high concentration in the blood of some infected swine, and they recede rather rapidly in others This is particu larly so if titer trends are compared with those of cattle It is not uncommon to find some infected swine with agglutinin titers at the 1 25 or 1 50 level and an occasional one with the agglutinin titer below 1 25 The foregoing situations, however, are usually associated with either early de velopment of infection and an ascending

titer or recovery from infection and a descending titer It has been the experience of the writer (Manthei, 1957) that only a very few swine that show clinical evidence of *Br suis* infection or have localized in fection without clinical manifestation fail to develop an agglutinin titer of 1 100 or higher at some stage of the disease Con sequently, frequent testing will partially eliminate the limiting factors of the sero agglutination test

Regardless of the foregoing limitations of the test, it has proved to be a valuable diagnostic test of herd infection There are very few herds infected with *Br suis* that do not contain some swine with agglutinin titers of 1 100 or higher This type of in formation is very significant, because it provides a sound basis for application of a control or an eradication method most suitable to a specific type of husbandry operation within each herd

Another limitation of the seroagglutu nation test and probably the one that causes the greatest interference with ac curate interpretations of reactions is the presence of nonspecific antibodies in the blood of noninfected swine Nonspecific agglutinins are most frequently observed in the 1 50 or lower dilutions of sera, but occasionally occur in sufficient concentra tion to be demonstrable in the 1 100 or higher dilutions Some nonspecific ag glutinins that cannot be differentiated from brucellar agglutinins by the standard ag glutination tests are believed to be pro duced by bacteria other than Brucella It has been demonstrated that some strains of other genera of bacteria and strains of the genus Brucella possess one or more common antigens (Francis and Evans 1926, Huddleson, 1943)

Since the limitations of the standard seroagglutination test are known, it be comes a matter of interpretation of the test Some of the early work (Cameron 1913) suggested that a titer of 1 25 in swine was evidence of exposure to Brucella but when subsequent studies (Cameron and Carlson, 1944, Crawford and Manthei 1948, Hutchings, 1944) were expanded

controlling diseases in animals always must be given full consideration

IMMUNITY

Ability of swine to resist infection caused by any of the three species of Brucella will be discussed from the aspects of natural resistance and acquired immunity

Nearly everyone who has investigated the problem of swine brucellosis has recognized a natural resistance in some swine of both sexes and of all ages to Brucella infections Manthei et al (1952) found that a majority of pigs nursed by dams with subacute or chronic infection were relatively resistant to Br suis prior to 12 weeks of age, but this resistance gradually decreased after that age The highest incidence of bacteremia and maximum blood titers occurred in these pigs shortly after weaning Goode et al (1952) reported that most pigs nursed by dams with acute brucellosis were resistant to Br suis prior to 4 weeks of age and this resistance gradually decreased up to 10 or 12 weeks of age In two experiments, Br suis was isolated from the tissues of only 2 of 44 boars and none of 57 sows after they were 6 months of age This evidence along with the decline in titers suggests that a large majority of the pigs had sufficient resistance either to prevent establishment of the disease or to recover from the primary infection These results are similar to those obtained by other earlier investigators, except Thomsen (1934), who found infection persisted in a significant percentage of swine at one year of age Hutchings et al (1941) found that weanling pigs were readily infected at 12 weeks of age with large doses of Br suis by five different methods of exposure The significant point in this report is that no recoveries of Br suis were made from any of the animals at the time of necropsy, which was 11 months following exposure This low recovery rate has not been observed by the writer in experimentally infected adult swine within a comparable period of time

Cameron et al (1910, 1911, 1913) reported that the majority of progeny obtained from mating of naturally resistant

boars and sows were highly resistant to oral administration of virulent Br suis

It has been the experience of the writer (Manthei, 1957) that approximately 30 per cent of all swine experimentally exposed show variable degrees of natural resistance to virulent Br suis This finding is similar to those of McNutt (1938) and Johnson et al (1931 1933) Moreover, swine show a higher degree of natural resistance to virulent Br melitensis and Br abortus than to virulent Br suis

The next point to consider is the ability of swine to acquire immunity to brucellosis from exposure to virulent Br suis According to Hutchings et al (1916b), swine previously exposed to virulent Br suis were less susceptible to a second exposure than unexposed swine of comparable ages were to their first exposure, but the acquired immunity was not sufficient to prevent reinfection Since there were no abortions in the 30 re exposed animals and only 3 isolations of Br suis at the time of parturition or necropsy, the principal criteria used for evidence of infection were bacteremia and increase of agglutinin titers These results are considerably different from those of the writer (Manthei, 1957), who was unable to reinfect more than 3 of 30 sows that showed evidence of brucellosis following a previous exposure Bacteremia was demonstrated in only 2 animals, agglutinin titers increased slightly followed by a rapid decline to the pre re exposure level, and Br suis was recovered from only 2 of 30 sows at time of parturition The greatest difference between the results of the two experiments is the incidence of post re exposure bacteremia Is a temporary bacteremia or a transient rise in titer conclusive evidence of brucellosis if one or both signs are the only manifestations of the disease? Since there were very few re exposed animals that showed clinical manifestations of brucellosis or from which Br suis was isolated at the time of parturition or necropsy, there is no evidence of multiplication of the infective agent in epithelial cells after it invaded the body of the host. The fact that manifestations of disease were considerably less apparent in

It would appear that both the allergic and seroagglutination tests employed have similar inherent deficiencies

Other aids to diagnosing brucellosis of swine are clinical manifestations and a record of addition of animals into the herd (Kernkamp, 1949) Although clinical manifestations of brucellosis are inconsistent and provide only presumptive evidence of infection, observing and reporting abnormal conditions frequently result in an early diagnosis before the disease becomes widespread Observance of clinical manifestations following addition of new animals or return of animals from public expositions is sufficient reason for one to be suspicious of brucellosis

There are very few diseases of swine that could be considered similar to brucellosis The principal exception probably is leptospirosis Both *Br suis* and *Leptospira pomona* cause a high percentage of abortions or stillbirths in susceptible herds, and the disease caused by each agent has a tendency to be self limiting Fever is usually present in the early stages of leptospirosis and absent in brucellosis The two diseases can be differentiated serologically *Brucella suis* can be isolated readily on direct culture, whereas *L pomona* is more difficult to isolate in this manner Unlike *Br suis*, *L pomona* does not produce clinical orchitis A distinct gross and histopathological lesion of chronic leptospirosis is a focal interstitial nephritis Spondylitis is most frequently associated with brucellosis and tuberculosis It is extremely difficult to differentiate the two types of spondylitis except by isolation of the causative agent

TREATMENT

There are no medicaments that have proved consistently effective in curing swine brucellosis (Crawford and Manthei, 1948) Numerous products have been used to treat human beings and small laboratory animals infected with *Brucella*, but this discussion will be limited to their use in swine Success with these products has been variable, depending on criteria used to

make the evaluation Bunnell *et al* (1953a) reported that treatment of *Br suis* infected swine with Aureomycin did not alter the agglutinin response, but it decreased the number of *Br suis* recoveries from routine collections of blood and from tissues at the time of necropsy below that of the untreated controls Hutchings *et al* (1950a) reported that a combined treatment of naturally and artificially infected swine with streptomycin and sulfadiazine caused a cessation of bacteremia and a diminution of the number of isolations at necropsy It was pointed out, however, that *Br suis* was isolated from tissues of 10 of the 15 treated animals at necropsy This indicates that the treatment had a marked bacteriostatic effect on the organisms circulating in the blood, but it did not prevent generalization or localization of infection In addition, the condition of treated swine was much poorer than that of untreated controls Cameron (1951) reported on feeding Aureomycin and vitamin B_{12} in an Aureomycin fermentation residue, to a group of pigs experimentally infected with *Br suis*, for 28 consecutive days Necropsies were performed immediately following the treatment period, and the organs were cultured and inoculated into guinea pigs *Brucella suis* was isolated from 1 of 14 treated animals and 8 of 10 untreated controls Although the results suggest that Aureomycin was bactericidal for *Br suis*, evidence obtained from studies on infected cattle at Beltsville indicate that this antibiotic may be only bacteriostatic No isolations of Brucella were made from milk of cows within 7 days after treatment was stopped, however, repeated isolations were made subsequent to that time

In summarizing the limited studies on therapy of brucellosis of swine, the medicaments employed were relatively ineffective in ridding the body of *Br suis*, but they produced a measurable bacteriostatic effect These discouraging results do not necessarily suggest that an effective treatment may not be developed, but the economic soundness of such an approach to

mitted from sows with genital infection to susceptible boars by repeated services under experimental conditions, boars prob ably become infected through a combi nation of oral and genital exposure under normal conditions Swine can be readily infected by conjunctival and intranasal exposure with a suspension of *Br suis* This experimental evidence suggests that swine could become infected with heavily contaminated aerosols and dust through the mucous membranes of the eyes and upper respiratory tract

Since it has been conclusively demon strated that susceptibility of swine of all ages and of both sexes varies considerably it would be difficult to assess the degree of influence that various transmission factors have on susceptibility The reason for this is that other factors such as status of preg nancy, size of exposure dose, species of Brucella involved, and virulence of the in vading organisms are indirectly related to susceptibility, and therefore affect the course of the disease

Experimentally, all three species of Bru cella are much more resistant to environ mental changes in a dried than in a moist state (Manthei, 1957) The survival rate of Brucella decreases as the temperature in creases regardless of their physical status (Carpenter and Boak, 1931, Manthei, 1957) Boak and Carpenter (1928, 1931a 1931b) reported that *Br melitensis* and *Br abortus* were killed at 140° to 142° F in 15 minutes, *Br suis* was not killed com pletely at 140° F in 20 minutes or at 142° F in 15 minutes, but all three species were killed at 145° F in 10 minutes Some of the most significant information on survival of Brucella has been obtained from experiments designed to simulate natural conditions Cameron (1932) demonstrated that *Br abortus* survived the longest in the dry state and at low temper atures on burlap sacking, soil, and bovine feces Kuzdas and Morse (1954) reported that *Br abortus* survived much longer in bovine urine, lake water, tap water, raw milk, bovine feces, and two types of soil at 2.5° C than at 37° C Freezing or near freezing temperatures permitted survival

of the same bacteria for at least 824 days Huddleson (1943) held hog spleens nat urally infected with *Br suis* at a temper ature of −10° F and in meat curing brine, and positive cultures were obtained after periods of 30 days and 40 days, respectively Brucella are destroyed in 2 to 4 hours when exposed to direct sunlight (Huddleson, 1943) Other results have been reported on the stability of Brucella under various environmental conditions by other investi gators

The rate of dissociation increases if Bru cella are maintained for a prolonged period at temperatures between 25° and 37° C in a medium that will support growth Nu merous other experimentally induced fac tors either retard or enhance dissociation of Brucella These results are significant if any of the experimental conditions are duplicated in nature, because dissociated Brucella are less pathogenic than their pro totypes

The use of disinfectants for destroying Brucella organisms, particularly in the presence of organic matter is a controver sial issue Romo (1941–42) reported that lye was the least affected when used in the presence of organic matter and a 2 per cent solution destroyed *Br abortus* Cresylic acid liquor cresolis compositus and so dium orthophenylphenate also destroyed *Br abortus* when used according to recom mended procedures The efficacy of all dis infectants that are bactericidal for Brucella is greatly enhanced if preceded by thorough scrubbing of contaminated sur face with large quantities of water This results in a marked dilution of the infec tive agent, the significance of which must be acknowledged in reducing the degree of exposure and thus reducing the chance of spread

Before discussing methods of control and eradication, this would appear to be the time to mention the importance of sani tation as a preventive measure (Crawford and Manthei, 1948, Hutchings, 1943, Man thei *et al*, 1956) Any type of procedure employed to improve sanitation will de crease the amount of contamination and indirectly decrease the possibility of ex

the re exposed than in the initially ex posed swine suggests that some unde termined protective mechanism prevented multiplication and localization of *Br suis* in epithelial cells of organs of the body

Although studies by a number of re search workers on the relationship of the various components of the blood and body tissues to natural resistance and acquired immunity have contributed much toward understanding the subject, our knowledge of the mechanisms of resistance and im munity is relatively incomplete

This brings us to the part of the discus sion that deals with immunity induced by vaccination McNutt and Leith (1943) vaccinated gilts between 3 and 5 months of age with a virulent *Br suis* culture They were given 2 ml subcutaneously and 5 ml intranasally All gilts ceased to react to the agglutination test prior to breeding Post vaccinal exposure was with the same strain of *Br suis* as the one used for vac cination All of the controls and 3 of the 12 vaccinated gilts aborted The remain ing 9 farrowed normal litters and otherwise did not show any infection Hadley and Beach (1922) also stated that vaccination of open gilts with a live culture of Brucella (porcine origin) conferred protection against infection

Within the past 15 years several investi gators have studied the immunizing value of Strain 19 against infection in swine caused by virulent *Br suis* With the ex ception of Holm et al (1945), all of them (Hoerlein, 1954, Jurado, 1950, Kernkamp and Roepke, 1948a, Lindley and Lander, 1949, Manthei, 1948) agree that Strain 19 vaccine will not produce a serviceable im munity against *Br suis* infection of swine

The writer (Manthei, 1948) conducted two experiments to determine the immuno genic properties of a *Br suis* strain of re duced virulence that originated in Aus tralia and is identified as King 8 Vacci nation of 4 to 6 month old gilts with a living vaccine prepared from this strain produced a serviceable immunity for 9 months but not for 24 months

Bunnell et al (1953b) reported that phenol and ether extract of *Br suis* cells

used to vaccinate swine did not protect the swine against subsequent exposure to virulent *Br suis* They also reported that a vaccine prepared from a mucoid phase of *Br suis* failed to protect swine against subsequent exposure to virulent *Br suis*

There is considerable doubt among persons who have a thorough knowledge of brucellosis of swine that vaccination would enhance eradication of the disease The reason for this attitude is that there are very few herds which could not be free of the disease within one year, if the own ers adopted one of three programs most suitable to the type of husbandry practiced in their herds This is possible because of the prolific nature of swine and the tend ency of brucellosis to be self limiting in a relatively high percentage of herds

EPIZOOTIOLOGY AND CONTROL

All three species of Brucella are capable of entering the body of swine in many different ways The avenues of entrance are the alimentary, genital, and respiratory tracts, conjunctiva and skin Brucella organisms gain entrance to the blood stream by penetrating the mucous mem branes of the different avenues of entrance The only exception would be entrance through denuded or possibly intact skin

Most of the experimental and field evi dence indicates that the majority of natural *Br suis* infection takes place through the alimentary and genital tracts The habits of swine and usual character of the disease strongly suggest that the alimentary tract is the most common mode of entrance for Brucella Moreover, the opportunity for suckling pigs to be exposed to *Br suis* when nursed by infected dams, for swine of all ages to eat food and drink water that has been contaminated with discharges from the genital tract urine, or feces, and for breeding stock to eat aborted fetuses and fetal membranes is excellent The next most likely natural avenue of entrance of *Br suis* is the genital tract. Sows and gilts are readily infected when bred to boars with genital infection or when arti ficially inseminated with semen containing *Br suis* Although brucellosis can be trans

4 If the herd is not readily freed of infection, abandon this plan in favor of Plan 1 or Plan 2

C Accessory Regulations

1 Blood samples are to be taken by an approved accredited Veterinarian

2 Reacting animals must be sold for immediate slaughter

3 Replacement swine may be added without test if procured directly from a certified brucellosis free herd

4 All other replacement breeding animals shall have passed a negative agglutination test and be held in isolation until passing a second negative agglutination test The second test shall be at least 30 days after the first, in the case of boars and open gilts, or after farrowing in the case of bred sows and gilts

5 All swine brought onto the farm for feeding purposes shall be segregated from the breeding herd until moved for slaughter

The Committee further recommends that

1 The present recommendation of the United States Livestock Sanitary Association for certification periods be increased from the present period of 6 months to a period of certification for one year

2 It is further recommended that all states adopt requirements for exhibition and the interstate movement of breeding swine as a step in the control of swine brucellosis

REFERENCES

AMARASINGHE, A 1955 New procedures for differentiating specific from nonspecific agglutination in bovine brucellosis serum and milk tests Extrait Arch Inst Pasteur de Tunis 2 259
—— 1955 New procedures for differentiating specific from nonspecific agglutination in bovine brucellosis serum and milk tests II A differential test for separating the nonspecific from the specific agglutination in suspicious titered serums Extrait Arch Inst Pasteur de Tunis 3–4 491

ANDERSON, W A AND DAVIS, C L 1957 Nodular splenitis in swine associated with brucellosis Submitted for publication in Jour Amer Vet Med Assn

ANDREWS F N AND HUTCHINGS L M 1946 Studies on brucellosis in swine IV Semen quality in Brucella infected boars Amer Jour Vet Res 7 38.

ARDREY, W B 1941 A study of factors influencing the isolation cultivation and differentiation of the species of Brucella Mich Agr Exp Sta Bull 177 1

BAY, W W WASHKO, F V BUNNELL DORIS E AND HUTCHINGS L M 191 Studies on the pathogenicity of Brucella abortus for swine II Proc Book 88th Ann Meet Amer Vet Med Assn

BENDTSEN H CHRISTIANSEN, M, AND THOMSEN A 19.1 Brucella enzootics in swine herds in Denmark — presumably with hare as source of infection Nord Vet Med 6 11
—— 1956 Brucella suis infection in hares as the cause of enzootic brucellosis in pigs Nord Vet Med 8 1

BENNETT G R 1941 Comparison of the agglutinability of several Brucella antigens (rapid test) Vet Med 12 455

BIRMAN, D T AND IRWIN M R 1948 Further studies of the bactericidal action of bovine serum for Brucella Proc 48th Meet Soc Amer Bact p 38

BOAK, R AND CARPENTER, C M 1928 The thermal death point of Brucella abortus in milk Jour Infect Dis 4 327
—— AND —— 1930 Brucella abortus agglutinins in porcine blood Jour Infect Dis 16 425
—— AND —— 1931a The thermal death point of Bacterium abortus in milk Jour Infect Dis 43 327
—— AND —— 1931b Lethal temperatures for porcine strains of Brucella abortus, with special reference to pasteurization Jour Infect Dis 49 485

BORTS, I H, McNUTT S H AND JORDAN, C F 1946 Brucella melitensis isolated from swine tissues in Iowa Jour Amer Med Assn 130 72.

BOYD, W L KERNKAMP, H C H ROEFKE, M H, AND BLIF, C L 1912. Incidence of brucellosis in swine Proc 38th Ann Meet. U S Livestock San Assn

BRAUDE, A I 1951 Studies in the pathology and pathogenesis of experimental brucellosis I A comparison of the pathogenicity of Brucella abortus, Brucella melitensis and Brucella suis for the guinea pig Jour Infect. Dis. 89 76
—— AND ANDERSON, D 1950 The pathogenesis and pathology of experimental brucellosis In Brucellosis. Waverly Press Inc, Baltimore p 50

BRAUN, W 1946 Dissociation in Brucella abortus A demonstration of the role of inherent and environmental factors in bacterial variation Jour Bact 51 327

posure of swine to Brucella organisms
The simple processes of scrubbing and
washing pens, feeding platforms, transpor
tation equipment, and rubber wearing ap
parel, burning or composting contaminated
litter, and burning or burying of aborted
fetuses and fetal membranes are examples
of things that any owner of swine can do
Other preventive measures are to purchase
swine from a Brucella free source, to quar
antine purchased swine of unknown source
until their brucellosis status is determined,
to refrain from using a community boar
for breeding of sows, to quarantine all
animals that are removed from and re
turned to the premises until owner is cer
tain they are free of brucellosis, and to be
certain that all swine entering a Brucella
free herd are transported in carriers that
have been thoroughly cleaned and disin
fected

There are three plans that generally have
been suggested or applied by research
workers and sanitary officials for the con
trol and eradication of brucellosis of swine
Since their recommendations have been
adequately discussed in the 1949 and 1954
Proceedings of the United States Livestock
Sanitary Association, they will not be dis
cussed in detail in this chapter Instead,
only a brief summary of plans of control
recommended by the Swine Brucellosis
Committee (Hay *et al*, 1954) of the
United States Livestock Sanitary Associ
ation in 1954 is presented

A Certification of Swine Herds as Bru
  cellosis Free

  Certification is made on the basis of two
  negative tests on the entire herd 30 to 90
  days apart This includes all animals 6
  months of age and over with no agglu
  tination tests being positive 1 100 or
  higher This certification is valid for 12
  months Recertification is made an
  nually by the passing of a single nega
  tive test on the entire herd

B Plans of Control for Infected Herds

  PLAN 1 Recommended for commercial
  herds

    1 Market the entire herd of swine for
      slaughter

    2 Clean and disinfect houses and

equipment Rest hog lots if pos
sible

3 Replace with stock from certified
  brucellosis free herds, preferably
  placing them on clean ground for
  as long as possible

4 Following two consecutive negative
  tests 30 to 90 days apart, the herd
  is eligible for certification

PLAN 2 Recommended for use in pure
bred herds where it is desirable to
retain valuable blood lines

1 Separate pigs from sows at 56 days
  of age or younger and isolate as
  completely as possible

2 Market infected herd as soon as
  practicable If sows are held for
  later litters, complete isolation is
  essential The disease has a greater
  tendency to spread as swine ap
  proach sexual maturity

3 Test the gilts to be used for the fol
  lowing breeding season about 30
  days before breeding Save only
  those gilts which are negative
  Breed only to negative boars

4 Retest the gilts after farrowing and
  before removing them from individ
  ual farrowing pens Should re
  actors be found, they should be
  segregated as far as possible from
  the remainder of the herd Only
  pigs from negative sows should be
  selected for breeding gilts

5 If herd is not clean at this time,
  the process is repeated another year
  As soon as the entire herd can pass
  two negative tests between 30 and
  90 days apart, it becomes eligible
  for certification

PLAN 3 Not recommended in general
but has been found useful in small
herds where only a few reactors are
found and where no clinical symp
toms of brucellosis have been noted

1 Remove reactors from farm

2 Retest herd at 30 day intervals re
  moving reactors, until entire herd
  is negative

3 Two clean tests, between 30 and 90
  days apart, qualifies the herd for
  certification

DONHAM, C R, AND FITCH C P 1933 A method for the preparation of antigen for the rapid agglutination test for Bangs disease with a technic for conducting such test Jour Amer Vet Med Assn 5 913

―――――― AND ―――――― 1936 Observations pertaining to standards of interpretation of agglutination titres in the diagnosis of Bangs disease Jour Amer Vet Med Assn 2 176

―――――― AND ―――――― 1936 An important factor in the mechanism of specific bacterial agglutination Jour Infect Dis 59 6

DOYLE, L P AND SPRAY, R S 1920 Infectious abortion in swine Jour Infect Dis 2 165

EICHHORN, A, AND THALLER, H I 1939 Preparation of Brucella antigen Proc 43rd Ann Meet of U S Livestock San Assn p 55

ELBERG S S, AND SILVERMAN, S J 1950 Immunology of brucellosis In Brucellosis Waverly Press Inc, Baltimore p 62

ELDER, C 1934 The transmission of Bang abortion infection from swine to cattle Mo Agr Exp Sta Bull 310 79

―――――― 1936 Transmission of Bang abortion infection of swine to cattle and the significance of low agglutination reactions in unbred gilts Mo Agr Exp Sta Bull 370 81

―――――― 1946 Transmission of Brucella suis from swine to cattle under pasture conditions Mo Agr Exp Sta Bull 398

EMMEL, M W 1930 An outbreak of Brucella disease in fowl Jour Amer Vet Med Assn 76 561

―――――― 1930 Susceptibility of turkey pigeon pheasant duck and goose to Brucella disease Jour Amer Vet Med Assn 77 185

―――――― AND HUDDLESON, I F 1929 Abortion disease in fowl Jour Amer Vet Med Assn 75 578

EVANS, A C 1918 Further studies on Bacterium abortus and related bacteria Jour Infect Dis 22 580

―――――― 1923 The serological classification of Brucella melitensis from human bovine caprine porcine and equine sources Pub Health Rep 38 1948

FABYAN, M 1912 A contribution to the pathogenesis of B abortus, Bang II Jour Med Res 21 441

FELDMAN, W H BOLLMAN J L AND OLSON, C JR 1935 Experimental brucellosis in dogs Jour Infect Dis 56 321

―――――― AND OLSON, C JR 1933 Spondylitis of swine associated with bacteria of the Brucella group Arch Path 16 195

FELSENFELD, O, YOUNG, VIOLA MAE, LOEFFLER, E ISHIHARA SACHIKO JANET, AND SCHROEDER W F 1951 A study of the nature of brucellosis in chickens Amer Jour Vet Res 12 48

FITCH, C P BISHOP, LUCILLE, AND BOYD, W L 1932 Report of further work on the relation of Bact abortus Bang to fistula and poll evil of horses Jour Amer Vet Med Assn 80 69

―――――― AND DONHAM, C R 1933 A discussion of some fundamental principles and practices underlying the application of the agglutination tests for Bangs disease Jour Amer Vet Med Assn 1 46

―――――― ―――――― AND BISHOP, L 1930 Monovalent and polyvalent antigens for use in the diagnosis of Bangs disease Proc Soc Exper Biol and Med p 553

―――――― ―――――― AND BOYD, W L 1930 Studies of the test tube agglutination test for the diagnosis of Bangs disease (contagious abortion) Minn Agr Exp Sta Bull 73

―――――― AND THOMPSON, CHARLOTTE M 1936 Studies of physical properties and agglutinability of Br abortus plate antigens from several sources Cornell Vet 3 222

FRANCIS, E AND EVANS, ALICE C 1926 Agglutination cross agglutination and agglutination absorption in tularemia Pub Health Rep 41

FRITZSCHE, K 1956 Brucellosis in hares in Germany Berl Munch tierarztl Wschr 16 301

GEE, L L, AND GERHARDT, P 1946 Brucella suis in aerated broth culture II Aeration studies Jour Bact 3 271

GILMAN, H L MILLS C H AND BIRCH, R R 1934 Passage of bovine Brucella through swine Jour Infect Dis 54 171

GLASSMAN, H N, AND ELBERG, S J 1946 The growth of Brucella in aerated liquid cultures Jour Bact 4 423

GOOD, E S AND SMITH W V 1916 Bacillus abortus (Bang) as an etiological factor in infectious abortion in swine Jour Bact 1 415

GOODE, E R, AND MANTHEI, C A 1953 Brucellosis of swine Report of the Chief Bur Anim Ind p 55

―――――― BLAKE, G E AND AMERAULT, T E 1952 Brucella suis infection in suckling and weaning pigs II Jour Amer Vet Med Assn 121 456

GRAHAM, R BOUGHTON, I B AND TUNNICLIFF, E A 1924 The presence of Bact abortus (Bang) in the bulbo urethral glands and seminal vesicles of an actively infected boar Abst Bact p 22

―――――― ―――――― 1930 Studies on porcine infectious abortion Ill Agr Exp Sta Bull 343 177

―――――― AND MICHAEL V M 1935 Brucellosis in swine Ill Agr Exp Sta Circular 435

GWATKIN, R 1931 Brucella abortus agglutinins in the blood of sows slaughtered in Toronto Cornell Vet 21 77

BRAUN, W 1947 Bacterial dissociation A critical review of a phenomenon of bacterial varia
    tions Bact. Rev 2 75
———— 1950 Variation in the genus Brucella In Brucellosis Waverly Press Inc., Baltimore, p 26
BROWN, I W FORBUS, W D., AND KERBY, G P 1945 The reaction of the reticulo endothelial
    system in experimental and naturally acquired brucellosis in swine Amer Jour Path 2 205
BUCK, J M 1924 The differentiation of primary isolation of *Bacterium melitensis* from primary
    isolations of *Bacterium abortus* (bovine) by their cultural and atmospheric requirements
    Jour Agr Res 12 585
BUNNELL, DORIS E BAY, W W AND HUTCHINGS, L M 1953a Studies on Aureomycin therapy of
    brucellosis in swine Amer Jour Vet Res 14 160
———— 1953b Studies on the vaccination of swine against brucellosis Amer Jour
    Vet Res 12 585
CAMERON, H S 1932 The viability of *Brucella abortus* Cornell Vet 3 212
———— 1943 Brucellosis in swine The interpretation of low titer reactions in experimental and
    field infections Amer Jour Vet Res 4 169
———— 1946 Brucellosis of swine IV The unit segregation system of eradication Amer Jour
    Vet Res 7 21
———— 1947 Brucellosis eradication and its effect on production in a large swine herd Cornell
    Vet 37 55
———— 1948 Swine brucellosis Proc 52nd Ann Meet U S Livestock San Assn., p 140
———— 1951 The bactericidal action in vivo of aureomycin in an aureomycin fermentation resi
    due against *Brucella suis* Cornell Vet 41 110
———— AND CARLSON, A 1944 Brucellosis of swine II Eradication by blood test and segrega
    tion Amer Jour Vet Res 5 329
———— AND ———— 1944 Brucellosis in swine III Studies on the diagnostic titer in the individ
    ual Amer Jour Vet Res 5 333
———— GREGORY, P W AND HUGHES E H 1941 Studies on genetic resistance in swine to Bru
    cella infection II A bacteriological examination of resistant stock Cornell Vet 1 21
———— ———— 1943 Inherited resistance of brucellosis in inbred Berkshire swine Amer
    Jour Vet Res 4 587
———— HOLM, L W AND MEYER, M E 1952 Comparative metabolic studies on the genus Bru
    cella I Evidence of a urea cycle from glutamic acid metabolism Jour Bact 5 709
———— HUGHES, E H AND GREGORY P W 1940 Studies on genetic resistance in swine to Bru
    cella infection Preliminary Report Cornell Vet. 2 218
———— AND MEYER, M E 1954 The differential effect of basic fuchsin and thionin on *D Alanine*
    utilization by the genus Brucella Amer Jour Vet Res 15 472
CARLE, B N AND DEWHIRST, W H 1942 A method for bleeding swine Jour Amer Vet Med
    Assn 101 495
CARPENTER C M AND BOAK R A 1931 Lethal temperatures for *Br abortus* with special ref
    erence to pasteurization Jour Bact 21 54
CHRISTIANSEN, M AND THOMSEN, A 1936 A contribution to surveying of the spread of brucellosis
    in hares in Denmark Nord Vet Med 8 841
COBURN, D K 1935 Slow and rapid agglutination tests in the diagnosis of Bang's disease A
    study of certain factors affecting the efficiency of these diagnostic tests No Amer Vet 16 2
CONNOWAY, J W, DURANT, A J, AND NEUMANN, H G 1921 Infectious abortion in swine Mo
    Agr Exp Sta Bull 187
———— 1925 Infectious abortion and immunity in swine Mo Agr Exp Sta Bull
    228
COTTON, W E 1919 Abortion disease of cattle Jour Amer Vet Med Assn 55 504
———— 1931 The character and possible significance of the Bang abortion bacillus that attacks
    swine Jour Amer Med Assn 78 506
———— AND BUCK, J M 1932 *Brucella abortus* in the blood stream of swine No Amer Vet 13 35
———— ———— AND SMITH, H F 1938 Communicability of infectious abortion between swine
    and cattle U.S.D.A Tech Bull 629
CRAWFORD, A B, AND MANTHEI, C A 1948 Brucellosis of swine U.S.D.A Circular 781
CREECH, G I 1930 Report of the Chief, Bur Anim Ind
———— 1931 Report of the Chief, Bur Anim Ind
———— 1932 Report of the Chief, Bur Anim Ind
———— 1935 Organic lesions in swine and caused by *Brucella suis* Jour Amer Vet Med Assn
    86 211
———— 1936 *Brucella suis* infection of the brain of swine Jour Amer Vet Med Assn 89 581
CRUICKSHANK, J C 1918 A simple method for testing dye sensitivity of Brucella species Jour
    Path and Bact 60 328
DAMON S R AND SCRUGGS, J H 1950 Recovery of *Br melitensis* from the hog Pub Health Rep
    65 574
DAVIS, C I 1957 A clinical case of brucellosis in a dog No Amer Vet 18 19
DILLZ, A L HUTCHINGS L M AND DONHAM, C R 1947 Studies on brucellosis in swine VI Clin
    ical and histologic features of intracutaneous reactions to fractions of *Brucella suis* Amer
    Jour Vet Res 8 225

———— Hasley, D E., and Torrey, J P 1927 Further studies on the isolation and cultivation of *Bacterium abortus* (Bang) Jour Infect Dis 2 352

———— Johnson, H W., and Hamann, E E 1933 A study of Brucella infection in swine and employees of packing houses Jour Amer Vet Med Assn 83 16

———— Richardson, M A., Warner, J., and Baltzer, B 1951 Studies in brucellosis III Mich Agr Exp Sta, p 5

———— Wood, E E, Cressman, A R., and Bennett G R 1945 The bactericidal action of bovine blood for Brucella and its possible significance Jour Bact 50 261

Hulse, E C 1952 Identification of the species of Brucella A comparative study of the different laboratory techniques employed FAO/WHO Expert Panel on Brucellosis WHO/Bruc./17

Hutchings L M 1943 Brucellosis in swine Proc 47th Ann Meet U S Livestock San Assn p 52

———— 1944 Report of further studies of brucellosis in swine Proc 48th Ann Meet U S Live stock San Assn p 103

———— 1947 Field control experiments with brucellosis of swine Proc 51st Ann Meet U S Livestock San Assn p 124

———— 1949 Swine Brucella control A comparison and contrast of control and eradication methods for swine and cattle brucellosis Proc 53rd Ann Meet U S Livestock San Assn p 43

———— 1950a Swine brucellosis *In* Brucellosis Waverly Press Inc Baltimore p 188

———— 1950b The natural course of swine brucellosis. 3rd Inter American Congress on Brucello sis, p 115

———— and Andrews, F N 1946 Studies on brucellosis in swine III Brucella infection in the boar Amer Jour Vet Res 7 379

———— Bunnell, Doris E., and Bay, W W 1950 Experimental therapy of brucellosis in swine with streptomycin and sulfadiazine Amer Jour Vet Res 11 388

———— Donham, C R, and Bay, W W 1950b The viability of *Brucella suis* in swine carcasses Proc Book 87th Ann Meet Amer Vet Med Assn, p 181

———— Delez, A L, and Donham, C R 1944 Studies on brucellosis of swine I Infection experiments with weanling pigs Amer Jour Vet Res 5 195

———— ————, 1946a Brucellosis in swine V Reproduction studies with naturally infect ed sows and boars Amer Jour Vet Res 7 388

———— ————, 1946b Studies on brucellosis of swine II Exposure and re exposure experiments with *Brucella suis* Amer Jour Vet. Res. 7 11

———— McCullough, N B Donham, C R Eisele, C W, and Bunnell, Doris E. 1951 The viability of *Br melitensis* in naturally infected cured hams Pub Health Rep 66 1102

———— and Washko, F N 1947 Brucellosis in swine VII Field control experiments Jour Amer Vet Med Assn 110 171

Irwin, M R, and Beach, B A 1946 Differential bactericidal activity of bovine serum toward strains of *Brucella abortus* of high and low virulence Jour Agr Res 72 83

———— and Bell, F N 1936 Studies on the bactericidal action of bovine whole blood and serum towards *Brucella abortus* and *Brucella suis* Jour Infect. Dis. 58 15

———— and Ferguson, L C 1938 Increase of bactericidins in the serum of cattle following recovery from infection with *Brucella abortus* Proc Soc. Exper Biol and Med 38 151

James W A and Graham, R 1930 Porcine osteomyelitis pyemic arthritis and pyemic bursitis associated with *Brucella suis* (Traum) Jour Amer Vet Med Assn 77 774

Johnson, H W, and Huddleson, I F 1931 Natural Brucella infection of swine Amer Vet Med Assn 78 819

———— ————, and Hamann E E. 1933 Further studies on natural Brucella infection in swine Jour Amer Vet Med Assn 83 727

Jordan, C F, and Borts, I H 1946 Occurrence of *Brucella melitensis* in Iowa Jour Amer Med Assn 130 72

Jurado, F. R., Cedro, V C. F and Moran, B L. 1950 Vacunación de porcinos con *Brucella abortus* Cepa 19 Ministerio de Agricultura y Ganadería, República Argentina Publicación Miscelanea 327 1

Kirby, G P, Brown, I W., Jr, Margolis, G and Forbus, W D 1943 Bacteriological observations on experimental brucellosis in dogs and swine. Amer Jour Path 19 1009

Kernkamp, H C H 1919 Clinical diagnosis of brucellosis in swine Vet Med 44 5-9

———— and Roepke, M H 1918a Vaccination of pigs with *Brucella abortus* vaccine Stra n 19 Jour Amer Vet Med Assn. 113 504

———— and ————, 1948b The interpretation of low agglutination titers in the control of swine brucellosis. Amer Jour Vet. Res. 9 46

———— and Jasper, D E. 1946 Orchitis in swine due to *Brucella suis*. Jour Amer Vet Med Assn 108 215

King, R O C 1947 Notes on recent research work with *Br suis infection* Vet Bull 37 533

Kingsley, A T 1939 Porcine brucellous Vet Med 34 637

Kronawetter, F R., Lear, S A., and Metzler, H J.. 1951 Thermal death time survives of *Bruce a abortus* in milk Jour Dairy Sci 34 1231

Kumas, C D., and Morse, E V 1951 The survival of *Bruce a abortus*, *B S D 3 Stra n 234* under controlled conditions in nature Cornell Vet 44 216.

HADLEY F B AND BEACH, B A 1922 An experimental study of infectious abortions in swine Wis Agr Exp Sta Bull 55

HALL W H 1950 Bactericidal action of human blood against Brucella and its specific inhibition 3rd Inter American Congress on Brucellosis p 87

—— AND SPINK W W 1947 Studies of immunity in brucellosis The bactericidal action of human blood against Brucella Proc Amer Soc Clin Invest, Abst Jour Clin Invest 26 1183

HASSELTINE H E 1930 Human and animal brucellosis Jour Amer Vet Med Assn , 76 330

HAY, J R. GARRETT H U HOERLEIN A B MANTHEI C A ROSNER, L A, AND SHANNON, E R 1954 Report of swine brucellosis committee Proc 58th Ann Meet U S Livestock San Assn p 204

HAYES F M 1934 Brucella infection in swine Jour Amer Vet Med Assn 37 322

—— 1937 Porcine brucellosis Vet Med 32 112

—— AND PHIPPS, H 1922 Studies in swine abortion Jour Amer Vet Med Assn 60 435

—— AND TRAUM, J 1920 Preliminary report on abortion in swine caused by *B abortus* (Bang) No Amer Vet 1 58

HENRY B S 1933 Dissociation in the genus Brucella Jour Infect Dis 52 374

—— 1933 Differentiation of bovine and porcine strains of *Brucella abortus* based on dissociation Jour Infect Dis 52 403

—— AND TRAUM, J 1930 A comparison of factors influencing the agglutination test for *Brucella abortus* Jour Infect Dis 47 367

HESS W R 1953 Studies on a nonspecific Brucella agglutinating substance in bovine serum I The differentiation of the specific and nonspecific agglutinins by heat treatment Amer Jour Vet Res 14 192

—— 1953 Studies on a nonspecific Brucella agglutinating substance in bovine serum II Isolation and purification of the Brucella agglutinating substances Amer Jour Vet Res 14 195

—— AND ROEPKE, M H 1951 A nonspecific Brucella agglutinating substance in bovine serum Proc Soc Exper Biol and Med 77 469

HOERLEIN, A B 1952 Studies in swine brucellosis I The pathogenesis of artificial *Brucella melitensis* infection Amer Jour Vet Res 46 67

—— 1953a Criteria for evaluation of a swine brucellosis program Iowa State College Vet 1 17

—— 1953b Studies on swine brucellosis III The differentiation of specific and nonspecific agglutination titers Cornell Vet 43 28

—— HUBBARD E D, AND GETTY, R 1951 The procurement and handling of swine blood samples on the farm Jour Amer Vet Med Assn 896 357

—— —— LEITH T S, AND BIESTER, H E 1954 Swine brucellosis Vet Med Res Inst Iowa State College

—— AND LEITH T S 1952. *Swine brucellosis control in a herd with valuable blood lines* Vet Med 47 448

HOLM, G C ARDREY, W B, AND BEFSO, W M 1945 A vaccination program for the control of swine brucellosis Proc 49th Ann Meet U S Livestock San Assn , p 191

HOWARTH J A, AND HAYES F M 1931 Brucellosis in the swine herd of the University of California Jour Amer Vet Med Assn 78 850

HOYER, B H 1950 Some aspects of the physiology of Brucella organisms *In* Brucellosis Waverly Press Inc, Baltimore, p 9

HUBBARD, E D, AND HOERLEIN A B 1952 Studies on swine brucellosis II Control in farm herds Jour Amer Vet Med Assn 900 138

HUDDLESON, J F 1921 The susceptibility of swine to contagious abortion *Bact abortus* (Bang) Mich Agr Exp Sta Quart Bull 4 43

—— 1923 Further studies on the susceptibility of swine to bovine infectious abortion Mich Agr Exp Sta Quart Bull 6 25

—— 1929 The differentiation of the species of the genus Brucella Mich Agr Exp Sta Tech Bull 100 1

—— 1931 Differentiation of the species of the genus Brucella Amer Jour Pub Health 5 491

—— 1932 The diagnosis of Brucella infection in animals and man by rapid macroscopic agglutination Mich Agr Exp Sta Bull 123

—— 1941a Progress made in the study of brucellosis during the past 25 years Jour Amer Vet Med Assn 98 181

—— 1941b The presence of a capsule on Brucella cells Mich Agr Exp Sta Bull 177 11

—— 1942 Immunity in brucellosis Bact Rev 2 111

—— 1943 Brucellosis in Man and Animals, rev ed The Commonwealth Fund New York

—— 1951 A study of factors that influence the isolation and growth of Brucella in or on culture mediums Proc 58th Ann Meet U S Livestock San Assn , p 123

—— 1955 Biochemical and histopathological reactions in the evolution of bovine brucellosis Mich State Univ Centennial Symposium Rep p 27

—— AND ABELL, E 1928 Behavior of *Brucella melitensis* and *abortus* toward gentian violet Jour Infect Dis 1 81

—— AND EMMEL M W 1929 Pathogenicity of the genus Brucella in fowl Mich Agr Exp Sta Tech Bull 103

Report on First Session Joint FAO/WHO Expert Panel on Brucellosis 1951 World Hlth Org Tech Rep 37

Report on Second Session Joint FAO/WHO Expert Panel on Brucellosis 1953 World Hlth Org Tech Rep 67

Report of the Univ of Ill Agr Exp Sta 1923 Infectious abortion in swine Circular No 271

RODERICK, L M, KIMBALL, ALICE, McLEOD W M AND FRANK E R 1947 A study of equine fistulous withers and poll evil Kansas Agr Exp Sta Bull 63

ROEPKE, M H 1953 Nonspecific agglutination reactions for Brucella Proc 57th Ann Meet U S Livestock San Assn, p 117

ROMO, R R 1941–42 The relative efficiency of the more commonly recommended disinfectants on Brucella abortus Rep N Y State Vet College p 85

ROSE, J E 1955 A study of nonspecific Brucella agglutinins in bovine serum Ph D thesis Univ of Minn

⸺, AND ROEPKE, M H 1957 An acidified antigen for detection of nonspecific reactions in the plate agglutination test for bovine brucellosis Submitted for publication in Amer Jour Vet Res

RUIZ CASTANEDA, M 1947 Studies on the pathogenesis of brucellosis Proc Soc Exper Biol and Med 64 298

RUNNELLS, R A 1941 Brucellosis of Swine In Animal Pathology Iowa State College Press Ames Iowa, p 476

SAN CLEMENTE C L, AND HUDDLESON, I F 1943 Electrophoretic studies of the proteins of bo vine serums with respect to Brucella Mich Agr Exp Sta Bull 182 1

SMITH, H C 1944 Eradication of swine brucellosis in a college herd Vet Med 39 249

SMITH, T 1920 Strain of Bacillus abortus from swine Jour Exper Med p 671

SPINK, W W 1952 Some biologic and clinical problems related to intracellular parasitism in brucellosis New England Jour Med 247 603

⸺ AND ANDERSON DOROTHY 1954 Experimental studies on the significance of endotoxin in the pathogenesis of brucellosis Jour Clin Invest 4 540

STABLEFORTH, A W 1953 Standardization of techniques for the diagnosis of brucellosis IV Con greso Internacional de Higiene y Medicina Mediterraneas Barcelona

⸺ 1954 The international standard for anti Brucella abortus serum Bull World Hlth Org 10 927

STARR, L E 1932 Production of agglutinins for Brucella abortus in calves swine and rabbits by skin and mucous membrane contact Jour Amer Vet Med Assn 81 230

STEFFENS, M 1937 Ueber die Bruzellose des Schweines Zugleich ein Beitrag zur Intradermal Probe als Diagnostikum Deutsch tierarzl Wschr 45 355

STONE, W C 1943 Brucellosis in swine Cornell Vet 32 115

THALER, H I 1940 A survey of field results with standardized Brucella antigen Proc 44th Ann Meet U S Livestock San Assn p 88

THOMSEN, A 1934 Brucella Infection in Swine Studies From an Epizootic in Denmark 1929–1932 Leven and Munksgaard, Copenhagen

TRAUM, J 1914 Report of the Chief Bur Anim Ind U S D A p 30

⸺ AND HENRY, B S 1929 Discrepancies of the agglutination test in Brucella abortus infec tion in cattle Cornell Vet 2 105

VAN DER HOEDEN, J 1932 Over spontane en experimenteele brucellainfectie bij den Hond Tijdschr v Diergeneesk 59 1383

VICTOR, J, MIKA, L A AND GOODLOW R J 1955 Studies on mixed infections II Pathological effects of combined Brucella suis and Coxiella burnetii infection Amer Med Assn, Arch Path 60 240

WASHKO, F V BAY, W W DONHAM, C R AND HUTCHINGS, L M 1951 Studies on the patho genicity of Brucella abortus for swine I Amer Jour Vet Res 12 320

⸺ AND HUTCHINGS, L M 1951 Studies on the pathogenicity of Brucella suis for cattle II Amer Jour Vet Res 44 165

⸺ ⸺ AND DONHAM, C R 1948 Studies on the pathogenicity of Brucella suis for cat tle I Amer Jour Vet Res 9 312

WEETER H M 1923 Infectious abortion in domestic animals Jour Infect Dis 32 401

LEVINE N D AND GRAHAM R 1950 The incidence of swine brucellosis in Illinois Jour Amer Vet Med Assn 116 443

LINDLEY D C AND LANDER J R 1949 Observations on the use of *Br abortus* Strain 19 in swine—including multiple intradermal doses Jour Amer Vet Med Assn 115 359

McCULLOUGH N B EISELE C A AND PAVELCHECK EMMA 1949 Isolation of *Brucella abortus* from hogs Pub Health Rep 64 537

———— 1951 Survey of brucellosis in slaughtered hogs Pub Health Rep 66 205

McNUTT S H 1934 Brucella infection in swine Jour Amer Vet Med Assn 84 620

———— 1935 Incidence and importance of Brucella infection of swine in packing houses Jour Amer Vet Med Assn 86 183

———— 1938 Brucella infection in swine Proc 42nd Ann Meet U S Livestock San Assn p 90

————, AND LEITH T S 1943 Swine brucellosis Mich State College Vet 4 28

————, AND MURRAY C 1924 *Bacterium abortus* (Bang) isolated from the fetus of an aborting mare Jour Amer Vet Med Assn 65 215

————, AND PURWIN P 1930 Effect of the *Brucella* group of organisms on chickens Jour Amer Vet Med Assn 77 212

MANTHEI C A 1948 Research on swine brucellosis by the Bureau of Animal Industry (1941–47) Amer Jour Vet Res 9 40

———— 1957 Unpublished data

————, RUTTLER A K AND GOODE E R 1956 Brucellosis Yearbook of Agr U S D A p 502

————, MINGLE C K AND CARTER R W 1952 *Brucella suis* infection in suckling and weanling pigs I Jour Amer Vet Med Assn 908 373

MARGOLIS G FORBUS W D AND KERBY G P 1945 Reaction of the reticulo endothelial system in experimental brucellosis of dogs Amer Jour Path 21 753

———— ———— AND LIDE T N 1947 Glomerulonephritis occurring in experimental brucellosis in dogs Amer Jour Path 23 983

MERCHANT I A AND PACKER R A 1956 The genus Brucella *In* Veterinary Bacteriology and Virology 5th ed Iowa State College Press Ames Iowa p 377

MEYER K F 1943 Observations in the pathogenesis of undulant fever *In* Essays in Biology Univ of Calif Press Berkley Calif

———— 1950 What is known about immunity to brucellosis? Proc 54th Ann Meet U S Live stock San Assn p 87

MILKS C H 1930–31 A study of some factors that influence the agglutination test for Bang abortion disease in cattle Rep N Y State Vet College p 1

MOORE T AND MACKIE C 1915 Factors affecting the tube agglutination test for Bang s d sease Canad Jour Comp Med 9 192

MORSE E V ERLING H AND BEACH B A 1951 The bacteriological aspects of experimental brucellosis in dogs follow ng oral exposure II Effects of feeding Brucella infected milk to young dogs Amer Jour Vet. Res 12 324

————, KOWALCZYK T AND BEACH B A 1951 The bacteriologic aspects of experimental bru cellosis in dogs following oral exposure I Effect of feeding aborted fetuses and placentas to adult dogs Amer Jour Vet Res 12 219

MUNGER M 1941 A study of the leucocytic picture in brucellosis Mich Agr Exp Sta Bull 177 35

MURRAY C McNUTT S H AND PURWIN P 1931 The result of agglutination tests of blood from animals on farms where cases of undulant fever occur Jour Amer Vet. Med Assn 78 339

———— ———— 1932 The effect of pasteurization upon *Br melitensis* var suis Jour Amer Vet Med Assn 80 336

NYKA W 1918 Estudio histobacteriologico de las lesiones producida por *Brucela* en animals y hombres Primera Reunión Interamericana de la Brucelosis Mexico D F p 675

OLSON C JR AND FELDMAN W H 1936 Attempts to isolate *Brucella abortus* from the blood of dogs in experimental brucellosis Jour Amer Vet Med Assn 88 51

PENNELL R B 1950 The chemistry of Brucella organisms *In* Brucellosis Waverly Press Inc Baltimore p 37

PICKETT M J NELSON, E L HOYT, R E. AND EISENSTEIN B E. 1952 Speciation within the genus Brucella I Dye sensitivity of smooth Brucellae Jour Lab and Clin Med 40 200

PLANZ, J F AND HUDDLESON I F 1951 Brucella infection in a dog Jour Amer Vet Med Assn 79 251

RENOUX G 1952a A new method for differentiating the varieties of Brucella the action of sod ium diethyldithiocarbamate (DEDTC) FAO/WHO Expert 1 anel on Brucellosis WHO/Bruc/47

———— 1952b Identification of strains of Brucella comparative results of serological and other methods FAO/WHO Expert Panel on Brucellos s WHO/Bruc/61

———— 1954 Selective media for the isolation of Brucella and more especially of *Brucella mel itensis* from contaminated material FAO/WHO Expert Panel on Brucellosis WHO/Bruc/106

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Anthrax*

history of anthrax is intimately associated with the history of bacteriology infectious diseases since it was with rax that Robert Koch in 1877 first onstrated conclusively the role of microorganisms in the cause of disease centuries preceding this monumental ., however, an endemic disease affect principally the herbivorous animals been recognized and described Additional historical significance is attached to anthrax since it was Pasteur s with *Bacillus anthracis* which first onstrated experimentally the protective value of an attenuated culture of the active organism in artificial immunity disease

anthrax is primarily a disease of herbivorous animals, however, almost all species mammals are susceptible to some degree sheep, cattle, horses, and swine are the most commonly affected of the domestic animals Man is susceptible, but the ease in this species occurs only sporadi-

cally, ordinarily from an individual contact with infected animals or animal products

Swine are generally considered rather resistant to anthrax as compared to sheep and cattle which are considered highly susceptible Swine may become infected however, along with other species of farm animals and may become important as a reservoir of infection

The incidence of anthrax in swine in the United States has been reviewed by Stein and Van Ness (1955) The following figures for the deaths due to anthrax depict the low level of incidence of the disease in swine

1951	1,088
1952	1,614
1953	127
1954	123
1955	11

The relatively large figures recorded in 1951 and 1952 were a result of the incorporation in swine feed of imported bone meal which contained spores of *B anthracis*

ETIOLOGY OF ANTHRAX

Anthrax is caused by *B anthracis*, a large Gram positive, aerobic, spore forming, non motile rod The individual bacilli are 1–1 5μ in diameter and 3–8μ long When observed in tissue from an infected animal, the organisms are commonly seen in short chains surrounded by a well developed

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normal Death follows in many of the swine within 24 hours after the cervical edema is noticed It is not uncommon for swine to recover even in the absence of treatment The swelling may disappear gradually and complete recovery appears to occur however, some such animals may remain carriers of *B anthracis*

Intestinal anthrax is characterized by the presence of the predominant lesions in the intestinal mucosa and the adjoining lymph nodes *B anthracis* presumably enters the mucous membrane or the lymphoid tissue of the intestine directly from the contaminated feed Only small areas may be involved or in severe cases several feet of the intestine may be affected (Van Es 1937) The mucosa in the affected area is swollen dark red in color, and necrotic areas or ulcers may appear (Hutyra *et al*, 1938) The mesenteric lymph nodes become enlarged Peritonitis is often observed in severe cases

Clinical signs of intestinal anthrax are not obvious as are those in the pharyngeal form In severe cases an acute digestive disturbance may be evident with vomiting, complete loss of appetite, and diarrhea with bloody feces Death may follow in the most severely affected swine, however, recovery occurs in many affected with the milder forms (Brennan, 1953)

Intestinal anthrax has been reported only rarely in the United States Many cases may be unrecognized because of the usual practice of avoiding a complete necropsy of animals suspected of anthrax It is possible that some of the animals dying of pharyngeal anthrax may also have had lesions in the intestinal tract Brennan (1953) reported that intestinal anthrax was the most common form of the disease seen in a 1952 outbreak of anthrax in swine in England

Septicemic anthrax is the highly acute form which results from the entrance of *B anthracis* into the blood stream, followed by rapid reproduction of the organisms throughout the body Death frequently occurs in animals so affected without any period of illness being noticed by the owner Septicemia is the usual occurrence in cattle and sheep however in swine it is the uncommon form of the disease Presumably there is a degree of resistance in swine not found in cattle and sheep which tends to bring about localization in most animals Goldstein (1957) reported that of 30 swine examined at necropsy during the anthrax outbreak of 1952 in Ohio only three had an enlarged dark spleen so characteristically seen in cattle It is possible that young pigs develop septicemia more frequently than older swine

PATHOLOGICAL CHANGES

In the interest of controlling anthrax complete necropsy of animals is strongly discouraged As a result there is relatively little detailed information available on the lesions in swine Superficial examination of the cervical region has however aided in an understanding of the usual appearance of the tissues in pharyngeal anthrax The tonsils are usually covered with a fibrinous exudate, or extensive necrotic changes may be evident The pharyngeal mucosa is frequently inflamed and swollen The cervical region is edematous but otherwise no superficial lesions are evident Incision of the region reveals an extensive infiltration of the subcutaneous tissues with fluid which is usually straw colored but may appear pink or hemorrhagic The tissue, containing large amounts of fluid may appear to possess a gelatinous consistency

The mandibular and suprapharyngeal lymph glands are enlarged to several times their normal size The cut surface of the affected gland may vary in color from deep brick red to strawberry red In more chronic cases the color may be grayish yellow, indicative of necrotic changes in the gland

TRANSMISSION OF ANTHRAX

Anthrax is generally considered a soil borne infection in cattle, sheep and horses Animal to animal spread does not commonly occur, but rather, *B anthracis* is deposited in the soil by the infected animal

capsule (Fig. 18.1). Under suitable aerobic conditions spores, which are highly resistant to disinfectants, heat, and dessication, may be produced

B anthracis grows very luxuriantly on most common laboratory media. On blood agar plates colonies can usually be detected within 12 hours. After 24 hours at 37° C. the colonies have a "ground glass" appearance with irregular, wavy borders which give them the "medusa head" characteristic No hemolysis is produced on blood agar, this is useful in distinguishing the colonies from those of certain nonpathogenic species of the genus (Nordberg, 1953) The colony of *B. anthracis* growing on blood agar on primary isolation possesses a stickiness which can be readily detected by touching with the bacteriological loop The colonial growth tends to adhere to the loop and forms tenacious threads This characteristic is presumably due to the capsular material

FIG. 18.1—*Bacillus anthracis* in a lymph node smear from a hog dead of pharyngeal anthrax. Stained with alkaline methylene blue, the bacterial cells appear blue and the capsule a light pink.   X 1,500.

on the organisms and is lost after one or a few transfers on artificial media.

B. anthracis is the only known species in the genus which is pathogenic for mammals, except for *B. cereus* which may produce death in mice or guinea pigs (Brown et al., 1955). The bacteriological diagnosis is relatively simple, therefore, when large, aerobic bacilli are found in an animal dead of an acute disease. Biochemical reactions may be used, but because of variability in the reactions of various strains or species in the genus, the injection of the culture into experimental animals, usually mice or guinea pigs, is considered the best method of identification.

CLINICAL SIGNS OF ANTHRAX

There are three forms of anthrax which have been observed in swine: pharyngeal, intestinal, and septicemic. Apparently the usual portal of entry is the oral cavity, and invasion occurs in the tonsils or mucosa of the pharynx. In some cases the infection may remain localized in the lymph nodes of this region and the disease would be classified as pharyngeal. In other cases the organisms may pass into the intestinal tract where primary invasion may also occur. When *B anthracis* is not localized but gains access to the general circulation, the septicemic form of the disease develops

Pharyngeal anthrax represents an infection which is limited to the lymph glands of the pharyngeal and cervical regions of swine by the body defenses. *B. anthracis* reproduces in large numbers in the lymph and probably also in the lymph glands and adjoining tissue. A local inflammatory process, interference with the flow of lymph, and consequent edema result. The swelling may become so extensive that it interferes mechanically with respiration and ingestion of food or water.

The clinical signs commonly observed in pharyngeal anthrax are cervical edema and dyspnea. General depression, inappetence and vomiting are commonly seen. Fever, with temperatures to 107° F, may occur, but it is not consistent and in some affected swine the temperature may be sub-

a lot which might have been contaminated and he was advised not to use it He obtained other feed but left the few sacks back in the corner In the fall he decided this feed looked all right and gave it to the pigs A few days later anthrax appeared

The determination of the source of infection was not always as simple as might be indicated in the preceding paragraphs One such case occurred on a farm where a feeding steer was down," presumably from overeating The owner did emergency slaughter, put the carcass in a food locker plant and gave the offal to the pigs During the following week some of the pigs were off feed and one died The diagnosis of anthrax was confirmed in the laboratory but investigation failed to connect the disease in the pigs with the known sources of contaminated feed The possibility of anthrax in the sick steer was considered and B anthracis was easily demonstrated in dried blood scraped from the floor under the chilled beef sides and in the beef liver which had been sliced, packaged, and frozen in the locker plant The source of the infection in the steer was never determined

In another one of the few bovine cases of anthrax observed during the Ohio outbreak, the source of infection was originally obscure Investigation revealed that the cattle had received no feed containing the contaminated bone meal Further history was obtained and investigators of the Division of Animal Industry discovered that ground feed for the group of feeding cattle had been prepared in a feed mixer which had been used to mix hog feed Some of the swine given this batch of feed developed anthrax It was concluded then, that the residue from the swine feed mixture (a few pounds of feed ordinarily remained in the mixer) contained anthrax spores which contaminated the cattle feed

On all farms where anthrax was recognized, the premises were quarantined and the carcasses were either burned or buried deeply as directed by the Division of Animal Industry A careful survey was maintained for any indication of secondary outbreaks from soil contamination No

such cases were recognized until July 1956 when on a farm where swine anthrax had occurred in 1952, two cows in a herd of 20 died of anthrax Cows had been pastured in the lot at intervals during the four year period Just prior to the reappearance of the disease however a tree was uprooted in a wind storm It was strongly suspected but unproved that anthrax spores remaining from buried swine carcasses were brought to the surface by the roots of the tree

DIAGNOSIS

Anthrax should be suspected when swine show cervical edema and dyspnea However erysipelas or malignant edema due to Clostridium septicum may also provoke similar clinical signs In malignant edema which in swine has also been called paranthrax the edema will often be more prominent in the shoulders or axillary spaces The edematous fluid and enlarged cervical or mesenteric lymph glands is seen on necropsy are also very suggestive of anthrax A history of the type of feed products eaten by the affected swine is always of value

The accurate diagnosis of anthrax is very important and in most cases is dependent upon the isolation and identification of B anthracis For these reasons the methods which have been used and found satisfactory by the authors will be described In swine, lymph glands from the affected area – cervical in the pharyngeal or septicemic forms mesenteric in the intestinal form – are the tissues of choice for bacteriological examination The blood or any of the organs will contain B anthracis in the animal dead of septicemic anthrax

Microscopic Examination

Impression smears were made by touching the freshly cut surface of the lymph gland to a slide The preparation was fixed by gentle heat and stained with Loeffler's alkaline methylene blue for two minutes An alternate method which gave good results consisted of heating the slide at fixation until the tissue began to char

at the time of, or following, death Spores are formed by some of the organisms and these highly resistant bodies may remain viable for years even under adverse conditions Subsequently, the spores may be ingested by susceptible animals and anthrax may develop

Swine can presumably become infected in this manner however, because of the small number of spores likely to be picked up and because of the higher degree of resistance in swine it probably occurs only rarely Rather, anthrax in swine generally occurs following ingestion of feed which contains a large number of *B anthracis* or viable spores Swine which are permitted to eat the carcass of an animal dead of anthrax may consume large numbers of organisms and may therefore become infected The use of bone meal or other animal products containing spores of *B anthracis* in feed is the most common source of infection in swine

The role of feed contaminated with spores of *B anthracis* in the transmission of anthrax can be illustrated by a brief account of the 1952 outbreak which occurred in the middle western states This outbreak was unique in that the source of contamination was determined early and also, to our knowledge, represents the largest outbreak of anthrax from a single source

During February, 1952, sick swine were seen by a veterinarian in southern Ohio, and he was aware of the presence of something unusual Anthrax was suspected and tentative confirmation by microscopic examination was rendered within a few hours Within two days final confirmation of the diagnosis was available Suspected cases followed in rapid succession in widely separated areas Within a week after the first case of anthrax was recognized, the veterinarians in the Division of Animal Industry, Ohio Department of Agriculture, had collected sufficient information to point conclusively to feed as a source of the infection As additional reports were investigated, the feeds, although a number of different feed companies were involved, had one thing in common All contained

bone meal obtained from a company in Columbus, Ohio A shipment of 100 tons of raw bone meal, imported from Belgium had been received in the plant Part of this had been incorporated into a meat scrap concentrate which was sold to a large number of feed companies These companies, in turn, mixed this product into swine feed, so that many hundreds of tons of feed were involved and at this time were scattered throughout Ohio and adjoining states The authors, and subsequently other laboratory workers, isolated *B anthracis* from the raw bone meal or the bone meal meat scrap mixture In many attempts the organism was not isolated from the mixed feeds to which the bone meal had been added, probably because of the excessive numbers of other bacteria present in such feed

Immediate steps were taken by the Division of Animal Industry to prevent the further distribution of the contaminated feed The distributor of the bone meal ceased operation and attempted to recover all shipments of the contaminated product which had not yet been used All mixed feeds which were known to contain the contaminated bone meal were returned to or held by the companies Because of the absence of identification or dates it was not possible in many cases to know which sacks of feed were or were not contaminated

Between February 22 and October 26, 1952, anthrax was detected on 258 farms in 57 counties of Ohio A total of 384 swine and 19 cattle died of the disease Only one or two animals died on the majority of the farms, partly because of immediate removal of the contaminated feed and also due to the use of antibiotic therapy There was no evidence of spread of the infection from animal to animal on the infected farms

By May, 1952, most of the contaminated feed had been destroyed or returned to the dealers for reprocessing, and as a result the disease incidence dropped very promptly Occasional cases did continue to appear as for example, on one farm where the owner was told that his feed was from

the unopened carcasses of animals dead of anthrax, few spores are formed except at the body openings When the animal is opened for a complete necropsy or when carnivorous animals are permitted to eat the carcass, there is usually extensive spore formation as the heavily infected blood and viscera are exposed to the oxygen of the air The most productive control measures include the complete destruction of the carcasses of animals dead of anthrax by incineration or by deep burial

It is generally recommended, when an animal dies in the open that it be burned on the spot If the animal must be moved the carcass should be placed on a sled or other vehicle which can be thoroughly disinfected, and hauled, not dragged to an area for disposal When this is not possible deep burial can be used The carcass should be covered with lime and at least four feet of dirt When carefully completed, either of these methods will minimize the chances of transmission of the infection

A freshly prepared hot lye solution (five per cent sodium hydroxide) is the disinfectant of choice This preparation should be used to clean up the premises immediately after the carcasses of the dead animals have been destroyed Litter or other contaminated articles should be burned and the lye solution used to scrub the exposed surfaces in buildings which are possibly contaminated

Swine rarely, if ever, pick up a sufficient dosage of anthrax spores from the soil to cause infection, and consequently, the epizootiology of anthrax in swine differs from that in cattle, sheep, and horses The higher level of natural resistance in swine presumably accounts for the fact that a much larger dosage of *B anthracis* is required to produce infection Thus anthrax of swine is almost always associated with feed which is more or less heavily contaminated In most cases the contaminated feed contains products of animal origin some of which came from animals infected with *B anthracis* Allowing swine to eat the carcass of an animal which may have died of

anthrax is an obvious example As described above the outbreak in Ohio occurred as a result of imported raw bone meal being mixed in swine feed

Following the outbreaks of anthrax in the Midwest in 1952 which were conclusively traced to imported bone meal regulations were established which prohibit the importation of raw bone meal into the United States (Stein 1953) Bone meal processed by an acceptable steam treatment may be imported under these new regulations In addition to this federal regulation some states have laws pertaining to the operation of rendering plants and the use of animal products in feed These regulations have proved effective since the occurrence of anthrax in swine has been limited to only a few cases in the past three years

PREVENTION AND TREATMENT

Since Pasteur's early work on immunization of sheep against anthrax, there have been occasional reports on improved methods of protective immunization These methods have been applied in swine only occasionally probably because of the sporadic occurrence of the disease in these species Schlingman *et al* (1956) reported the use of an alum precipitated culture filtrate in cattle, sheep and swine The product increased the resistance of the immunized swine however, the results were somewhat inconclusive since the control animals were not uniformly infected by the challenge with *B anthracis* It is suggested however that immunization would reduce the incidence of infection when swine are exposed to massive doses of *B anthracis* Immunization of swine on a large scale has not been recommended however since swine possess a level of natural resistance adequate to prevent the disease except following heavy exposure to *B anthracis*

Treatment of animals infected with *B anthracis* is not commonly practiced In the more susceptible species the animal is frequently moribund or dead before the diagnosis is reached Since swine can de

gray in color The alkaline methylene blue was immediately placed on the slide and within 5 seconds was washed with water B anthracis appears blue in color while the capsule is stained a light pink Often the body of the bacillus stains faintly and unevenly, suggestive of disintegration (Fig 18 1)

Spores are not observed in slides prepared from fresh tissue or from freshly cut surfaces Spore forming anaerobes are frequently encountered in tissues of animals which have been dead several hours prior to necropsy Differentiation is important in such cases, and the following points are helpful Spores are rarely seen in B anthracis in fresh tissue preparations, while spores are regularly seen in clostridia In the latter organism the rod is usually enlarged somewhat by the spore Capsules are not observed on the clostridia

Cultural Studies

B anthracis grows readily on many common culture media and it is characterized by very rapid colonial development Typical colonies can be observed after 12 to 18 hours of incubation This rapid growth is useful in differentiating B anthracis from other pathogens If other non pathogenic bacilli are present, animal inoculation is essential to be certain of proper identification

B anthracis is readily cultivated from the enlarged lymph glands and it may also be demonstrated from the surrounding connective tissue in some cases In the occasional septicemic case the organisms can be isolated from the blood, spleen, liver — in fact, from essentially any tissue of the body Since B anthracis grows more rapidly than most of the saprophytic bacteria likely to be encountered, except other species of Bacillus, one should always examine the cultures after incubation of 12 to 18 hours

The clostridial species commonly found in swine, either as pathogens or from post mortem invasion, are strict anaerobes and therefore will not grow on regular aerobic cultures This simple procedure

avoids placing reliance on the microscopic examination of the tissue when one is uncertain about the identity of rod shaped organisms

During the latter part of the outbreak in Ohio in 1952, many deaths in swine were suspected to be due to anthrax but proved to be from other causes This undoubtedly resulted from the extensive publicity given to the disease and from the fact that whenever a hog died a veterinarian was consulted Under normal conditions the sporadic death would probably have gone unnoticed Many of these deaths were actually malignant edema A few cases of acute swine erysipelas were found and a few of the deaths were caused by salmonella infections

CONTROL

The control of the spread of anthrax differs significantly from the control of most of the other important animal diseases The highly resistant spore formed by B anthracis accounts for this difference Some swine may become inapparent carriers, but there is little evidence to indicate that this forms an important source of infection to susceptible animals Otherwise, animals which become infected do show clinical signs and generally develop an acute disease which terminates in death within a few days Transmission from animal to animal rarely occurs, but rather, soil contaminated by the organisms serves as a source from which susceptible animals subsequently ingest the spores Because of this common form of transmission, anthrax can be controlled by preventing susceptible animals from contacting viable spores of B anthracis

Van Ness and Stein (1956) pointed out the importance of soil types in the survival of anthrax spores The principal areas of enzootic anthrax are in regions characterized by soils high in nitrogen and with adequate calcium Where such soil types are lacking (central and eastern states) anthrax does not appear to persist

The spores can survive for years under a variety of environmental conditions In

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CHAPTER 19

Clostridial Infections

Those microorganisms included in the genus Clostridium are the spore-forming, anaerobic bacteria. The young or vegetative forms are rod-shaped, Gram-positive, and quite large. When spores are formed, they are spherical to ovoid and generally greater in diameter than the vegetative rod. The spores may be located centrally, sub-terminally, or terminally within the rod, but there is sometimes a tendency toward consistency of location within a given species; e.g., the spores of *Cl. tetani* tend to locate terminally, giving the so-called "drum-stick" or spoon-shaped appearance. Many, but not all, are motile by means of peritrichous flagella when cultures are young (Hagan and Bruner, 1957; Kelser and Schoening, 1948; Merchant and Packer, 1956).

The clostridia may be divided into two groups on the basis of their disease-producing potentialities. One group consists of those that have power to invade and multiply in tissues. They are sometimes referred to as the "gas gangrene" group. Many of them are found in wound infections; others, however, enter the body

via the digestive tract The second group shows little or no power of invasion but under the right conditions is capable of multiplying and producing extremely powerful toxins This group includes the organisms of tetanus and botulism Since, in the case of the latter, the toxin is believed to be chiefly pre-formed, or formed outside the animal body, and the disease is caused, under natural conditions, by ingestion of the toxin(s) rather than the organisms *per se*, botulism will be dealt with separately in Chapter 33 under Section V, Toxemias and Poisonings The remaining clostridial infections, as they affect swine, are here discussed under the headings of blackleg, malignant edema, and tetanus. Although one case of infectious hemoglobinuria caused by *Cl. hemolyticum* (Records and Huber, 1931) and an outbreak of hemorrhagic enteritis apparently due to *Cl. perfringens*, type C (Field and Gibson, 1955) have been described in swine, the writer feels that these conditions are not yet of sufficient importance in swine to warrant further emphasis at this time.

Blackleg

Blackleg is an acute, infectious disease associated with high mortality. It is principally a disease of cattle, less often of other ruminants, and rarely of swine. In cattle the disease has also been known as black

quarter, quarter ill, and symptomatic anthrax. Until recently, conclusive evidence of isolation of the blackleg organism from disease in swine has been lacking This fact, together with the difficulty

velop a more chronic form of the disease, treatment can be successfully administered in some cases. In the outbreak in Ohio in 1952, penicillin in oil was used at a dosage level of 10,000 units per pound of body weight. According to Goldstein (1957) pigs which were showing clinical signs of anthrax recovered completely after this treatment and the losses were reduced considerably when the disease was recognized early in its course. Anthrax antiserum in doses of 20–75 ml was also used in treatment of a limited number of animals. The results were comparable to those following treatment with penicillin in that the pigs which were in the early stages of anthrax recovered promptly.

CONCLUDING REMARKS

Anthrax in swine is a sporadic disease which occurs only when the animals have access to contaminated feed. Consumption of the carcass of an animal dead of anthrax or of feed containing contaminated animal products, such as bone meal, is the usual

history. However, anthrax in swine should be viewed as an important disease, since these relatively resistant animals may become carriers or may serve as a source of infection to the more susceptible species.

The type of soil appears to be important to the survival of *B. anthracis*, and in a favorable soil area swine may maintain the disease and spread it to new fields, thereby increasing the chances of infection of cattle and sheep.

Incineration or deep burial of all animals which die of anthrax and complete disinfection of the contaminated area will assist in reducing the incidence of this disease. The practice of permitting swine or other carnivorous animals access to the carcasses of animals dead of undiagnosed disease should be discouraged. Careful control of the processing of animal products for swine feed to insure destruction of anthrax spores and the importation of bone meal or other animal products only after proper processing to destroy any spores will greatly reduce, or eliminate, anthrax in swine.

REFERENCES

Brennan, A. D. J. 1953 Anthrax, with special reference to the recent outbreak in pigs. Vet Rec 65 255.

Brown, E. R., Cherry, W. B., Moody, M. D., and Gordon, M. A.: 1955 The induction of motility in *Bacillus subtilis* by means of bacteriophage lysates. Jour. Bact. 69 590.

Goldstein, H. 1957 Personal communication.

Hutyra, F., Marek, J., and Manninger, R. 1938 Pathology and Therapeutics of the Diseases of Domestic Animals, Vol 1, p 13. Alexander Eger, Chicago.

Nordberg, B. K. 1953. Continued investigations of some important characteristics in anthrax-like microorganisms as viewed from a point of view of differential diagnosis. Nord Vet Med 5 915.

Schlingman, A. S., Devlin, H. B., Wright, G. G., Maine, R. J., and Manning, M. C. 1956. Immunizing activity of alum precipitated protective antigen of *Bacillus anthracis* in cattle, sheep and swine. Amer. Jour. Vet. Res. 17.256.

Stein, C. D. 1952 Anthrax in the United States and its control. Proc. U. S. Livestock San Assn., p 67.

———. 1953. A review of anthrax in livestock during 1952 with reference to outbreaks in the first eight months of 1953. Proc. U. S. Livestock San. Assn., p. 101.

———. 1955 Anthrax in livestock during 1955. Vet. Med. 51.539.

———, and Van Ness, G. B.: 1955. A ten year survey of anthrax in livestock with special reference to outbreaks in 1954. Vet. Med. 50.579.

Van Es, L.: 1937. Anthrax in swine. Jour. Amer. Vet. Med. Assn. 90 331.

Van Ness, G., and Stein, C. D.: 1956. Soils of the United States favorable for anthrax. Jour. Amer. Vet. Med. Assn. 128.7.

cision of the affected area usually reveals serous or sero hemorrhagic exudate in the subcutaneous and intermuscular connective tissues with a varying but generally mild degree of gaseous admixture Incision of the muscles themselves shows, characteristically, darkening of the muscles and small cavitations or gas pockets within them The cut surface of the muscle is relatively dry, and the freshly incised tissue emits a butyric acid like or 'rancid butter' odor The contiguous lymph nodes may be swollen, reddened, and even emphysematous In addition to the localized lesion, there may be mild sero fibrinous effusions in the serous cavities, hepatic enlargement with or without the presence of visible gas bubbles, or both Evidence of septicemia may be present in the form of limited petechial or ecchymotic hemorrhages, particularly on the serous membranes The blood is dark red and clots well

Microscopically, the muscle lesions reveal necrosis, hyaline degeneration, cavitations due to the presence of gas bubbles, hemorrhage, and some edema and leukocytic infiltration Necrosis is prominent toward the center of the lesion, and leukocytic infiltrations are more marked toward the periphery

DIAGNOSIS

Blackleg must be differentiated particularly from anthrax, malignant edema, and other forms of cellulitis Diagnosis is based chiefly upon the results of pathogenicity and serum protection tests following isolation of the causative organism In most cases the localized lesions found at necropsy will suggest the alternatives of blackleg or malignant edema Most other forms of cellulitis will be characterized by purulent exudates Anthrax in swine tends to localize in the retropharyngeal or peripharyngeal areas causing respiratory distress through edema of the larynx Furthermore, the likelihood of anthrax or of blackleg, when present, is often suggested by the clinical history Involvement of the muscle tissue, with reddish to blackish discoloration, gas

pockets, and relative dryness of the cut surface, suggests blackleg Primary involvement of the connective tissues, with a more moist cut surface due to edema and hemorrhage suggests malignant edema However, since the variations in the lesions are often more quantitative than qualitative and since the organisms of both diseases are often found together in the same disease process, a reliable etiological diagnosis usually cannot be made upon the basis of the lesions alone but rather depends upon isolation of the causative organism(s) and the results of pathogenicity and/or protection tests Guinea pigs and rabbits appear to be the animals of choice for pathogenicity tests since the guinea pig is susceptible to both infections while the rabbit shows considerable resistance to *Cl chauvoei* The hamster also is reportedly very susceptible to the blackleg organism while the mouse is moderately susceptible Mice are useful in protection tests, being susceptible both to the organism of blackleg and to that of malignant edema and having the advantage of requiring small doses of culture and protective immune sera

TREATMENT

Until the advent of the antibiotics there was little hope of successful treatment of blackleg Aitken (1919) reported that penicillin is effective in cattle when used early Percival *et al* (1953) reported that Aureomycin is effective in sheep Here, too, the treatment should be given early Potent antiserum can be made, but its use in treatment of blackleg can hardly be justified when its cost and effectiveness are compared to those of the antibiotics Effective treatment of swine apparently has not yet been reported

IMMUNITY

The infrequent occurrence of blackleg in swine has failed to stimulate widespread attempts to immunize this species against the disease Since immunizing agents are effective in other animal species susceptible to the disease, there seems to be little room for doubt that pigs would respond to the

of infecting pigs artificially, has caused several writers (Meyer, 1915, Heller, 1920, Hutyra et al, 1949, Kolle and Wassermann, 1928 Glasser et al, 1950) to express the opinion that swine are immune Eggleston (1950) described a disease resembling blackleg in pigs in which an organism was isolated resembling that of blackleg morphologically and which was lethal for guinea pigs but not for rabbits Sterne and Edwards (1955) have described a disease resembling blackleg in swine from which the blackleg organism was isolated The pigs were being kept on premises where the practice of keeping cattle had been discontinued on account of severe losses from blackleg Gualandi (1955) reported an outbreak of *Cl chauvoei* infection causing death of 15 out of 34 pigs within a period of 2 days

The disease in cattle is seemingly found wherever the cattle industry is important Man apparently is immune Guinea pigs and mice are susceptible, rabbits and rats are relatively resistant

ETIOLOGY

Although Orfeur and Hebeler (1953) reported what appeared to be a typical outbreak of blackleg in pigs from which only *Clostridium septicum* was isolated, blackleg in other species, and presumably in pigs also, is caused by *Cl chauvoei* (*Cl feseri*) The organism has also been known as *Bacillus chauvoei, Bacillus feseri, Bacillus gangraenae emphysematose,* and *Bacillus anthracis symptomatici* The vegetative form is a large rod, 0 6–0 8μ wide by 3–8μ long with rounded ends It is seen singly, in pairs, and sometimes in short chains or in filaments Young cultures are motile by means of peritrichous flagella and are Gram positive, older cultures show less motility and take the Gram stain erratically

Clostridium chauvoei is a relatively strict anaerobe and grows best at 37° C It requires the addition of blood or tissue to most media for growth Liver or liver brain media with slightly alkaline pH give best results The organism is non proteolytic and liquefies gelatin only slowly and

when serum is incorporated Older cultures emit a butyric acid odor When anaerobiosis is complete, surface colonies can be grown on blood agar Hemolysis is slight Carbohydrates which are quite regularly fermented with the formation of acid and gas are glucose, galactose, lactose, levulose, maltose, and sucrose (Hagan and Bruner, 1957, Kelser and Schoening 1948 Merchant and Packer, 1956)

PATHOGENESIS AND CLINICAL SIGNS

Clostridium chauvoei apparently forms only a relatively weak exotoxin The exact part which it plays in pathogenesis is unknown, but it probably is not the most important lethal factor The organisms apparently can enter the body via wounds or the digestive tract Reaching the site of predilection, which is usually in striated muscle, the organisms multiply by fission and produce acute inflammation with considerable gas formation, fever, bacteremia, toxemia, and death The paucity of data on blackleg in swine precludes a full description of the symptoms in that species. Since the lesions resemble those in cattle it might be assumed that the symptoms would also follow a pattern similar to those in that species The presence of lameness when the heavily muscled portions of the body are involved, of a cold swelling with crepitation when gas is plentiful in the lesion, and of fever in all cases seems assured, as well as anorexia and rapidly progressive weakness and depression The disease runs its fatal course in 1 to 3 days

PATHOLOGICAL CHANGES

Localized swelling over one or more of the heavily muscled portions of the body may be so marked as to be obvious Crepitation may often be determined then as easily as before death or more so, since the organisms may continue to carry on their metabolism for some time after death However it may also be so small as to require careful search or even to escape detection The lesion is most likely to be encountered in the hind quarter, loin, or forequarter in

matis malignı II, *B oedematis thermophilus, B gigas*) or *Clostridium perfringens*
(*Cl welchii*) (Geiger, 1929) In fact, the
flora is sometimes multiple Certainly *Cl
septicum* is widely regarded as the most important cause, both in swine and in other
species It will be so regarded here

The vegetative forms of *Cl septicum* are
0 5–0 6μ by 2–8μ in size with rounded ends
and they occur singly or in chains or long
filaments Spores are oval and central or
subterminal Young cultures are motile
and Gram positive, but with age lose both
properties It is, perhaps, not as fastidious
in growth requirements as *Cl chauvoei*, re
quiring less strict anaerobiosis and less en
richment of media Deep agar colonies are
cotton like and filamentous Gelatin is
slowly liquefied Among the carbohydrates
usually fermented with production of acid
and gas are glucose, galactose, lactose, levu
lose, maltose, and salicin A toxin is pro
duced, the lethal portion of which is desig
nated as the alpha toxin and the hemolytic
portion as the beta toxin The toxin is
potent enough that filtrates of a 24 hour
culture, when injected intravenously in
0 5–1 0 ml doses, usually kill guinea pigs
or rabbits (Hagan and Bruner, 1957, Kel
ser and Schoening 1948 Merchant and
Packer, 1956)

PATHOGENESIS AND CLINICAL SIGNS

Mullally (1941) has likened the relation
ship of anaerobic infection with gas gan
grene to that of pyogenic bacteria with septicemia. That is, the bacterial infection may
often occur without the fully developed
sequel He states that *Cl septicum* and *Cl
perfringens (welchii)* are the most danger
ous of the spore bearing anaerobes from the
standpoint of gas gangrene He goes on to
state that masses of damaged or devitalized
tissue, especially when deeply situated or
confined in such a way that the products of
infection cannot escape, are conducive to
gas gangrene or malignant e d e m a
Wounded muscles are peculiarly disposed
to help provide the right conditions since
injury causes reflex contraction which pulls

the damaged fibers apart creating a cavity
Hemorrhage and swelling occur within the
muscle sheath, prolapse of muscle, fascia
and adipose tissue close the wound open
ing and a suitable environment for rapid
multiplication of the organism and for
mation of toxin is created Thus wounds
in the more heavily muscled areas are more
likely to develop malignant edema than
those in skin subcutis, bone, or even brain

Different strains of *Cl septicum* and *Cl
perfringens* vary in their abilities to pro
duce hyaluronidase Addition of testicular
hyaluronidase greatly alters the character
istics of non hyaluronidase producing
strains causing rapid extension of the
edema (McLean and Rogers 1943) Most
strains of these species produce an active
fibrinolytic substance (Reed *et al* 1941)
Both of these substances – if, indeed, they
are separate – probably play important
parts in spread of the toxin and the infec
tion

It is believed that death and probably
most of the other symptoms of malignant
edema are due to its toxin (s) In the case
of *Cl septicum*, the beta toxin, which has
hemolytic action, has been identified as
desoxyribonuclease (Warrack *et al*, 1951)
Kellaway *et al* (1941) investigated the cir
culatory effects of intravenous injection of
Cl septicum toxin in the cat and rabbit
Both species showed a specific effect on the
heart, resulting in a fall in systemic and
rise in venous blood pressures In isolated,
perfused hearts, both a direct action upon
the muscle and coronary vasoconstriction
were observed In both species a pul
monary vasoconstriction apparently played
a part in failure of the heart

Malignant edema is characterized locally
by soft to doughy swellings which develop
and spread rather rapidly There is usually
little or no detectable crepitation Location
or size of the swelling, or both, may inter
fere with locomotion General manifes
tations of the disease include fever, ano
rexia, weakness, depression, and death The
course of the disease is short, usually from
a few hours to 2 or 3 days.

available antigens In fact Sterne and Edwards (1955) report evidence which suggests that they do

The evolution of effective blackleg antigens started with those of Arloing *et al* (1887) and Kitt (1888) and progressed successively through the natural aggressin of Schobl (1910) and Franklin and Haslam (1916) and the artificial aggressin of Nitta (1918) to the bacterin of Leclanche and Vallee (1925) The bacterin has been used extensively in cattle in this country and has proved to be a safe, reliable, and relatively inexpensive immunizing agent The cultures are concentrated, formalized, and in some cases have alum added to them to retard absorption and enhance the antigenicity In view of the common coexistence of *Cl chauvoei* and *Cl septicum* in the same environment and even the same sick animal and the common necessity of animal inoculation tests for accurate diagnosis, it has become a common and successful practice to use bivalent bacterins made with these two organisms They also have been used in swine. *A highly potent antiserum can be made for protection against Cl chauvoei, but its use is seldom justified, especially in swine*

EPIZOOTIOLOGY AND CONTROL

Mercuric chloride and formalin are *probably the most effective of the chemical*
disinfectants against *Cl chauvoei* The spores are said to be killed by mercuric chloride in 1 500 solution in 10 minutes and by 3 per cent formalin in 15 minutes Coal tar disinfectants are also said to be effective In view of their susceptibility to disinfectants, it is perhaps surprising that the spores resist 100° C dry heat for several hours and boiling water for almost 2 hours

In spite of the resistance of *Cl chauvoei* spores to natural influences, special control measures for prevention of blackleg in swine are seldom needed, due apparently to a significant natural resistance to the infection It seems clear that pigs need not be immunized against blackleg as a routine measure If it becomes necessary to subject them to exposure, or to expose them to soil contaminated with blackleg spores within the previous 8 to 10 years, it would seem advisable first to protect them against the infection by using the *Cl chauvoei septicum* bacterin In its spore form, *Cl chauvoei* is extremely resistant and may retain its virulence in soil for several years It probably enters the body of the pig via the digestive tract However, it must be admitted that natural resistance might afford sufficient protection even against such exposure Administration of bacterin may be followed by stiffness for *18 hours (Eggleston, 1950)*

Malignant Edema

Malignant edema is an acute and usually fatal wound infection disease of animals, including swine Its broad array of susceptible hosts includes cattle, sheep, swine, horses, man, dogs, cats, guinea pigs, rabbits, mice, and pigeons Susceptibility varies from horses and sheep, which are highly susceptible, to dogs and cats, which rarely contract the disease under natural conditions In sheep it also causes a disease known as braxy, which is prevalent in northern Europe It is believed that the organism of this disease enters the body via the digestive tract since the primary location is the abomasum It is present in
the soil and is widely distributed over the world Whether it multiplies in the soil is unknown, but it has been thought to increase in soils rich in organic matter (Varnell, 1919)

ETIOLOGY

Malignant edema is generally regarded as due to *Clostridium septicum (Vibrion septique, Bacillus septicus, Cl or B oedematis maligni)*, and rightly so However diseases indistinguishable from malignant edema except by careful bacteriological studies are occasionally due also to *Clostridium novyi (Cl oedematiens, Bacillus oede*

EPIZOOTIOLOGY AND CONTROL

In other farm animals malignant edema is sometimes associated with parturition apparently through contamination of lacerations incurred during the process Geiger (1929) says that gas edema (presumably malignant edema since he regarded *Cl novyi*, *Cl septicum*, and *Cl welchii* as causes) in swine may originate in skin wounds of various kinds such as subcutaneous and intramuscular injections and tail bleedings, and that the local lesions occur close to the portal of entrance In two cases in which the lesions involved the stomach wall, the infection presumably entered via the mouth and wounds in the gastric mucosa In the writer s experience, nearly all cases have been associated with hog cholera immunization procedures Since we know that the causative organisms are widely distributed in the soil and are highly resistant to natural influences when in the spore form it logically follows that the most practicable and effective means of control is the use of proper sanitary techniques in all surgical and vaccinal procedures

Tetanus

Tetanus (lockjaw) is a wound infection disease characterized by intoxication accompanied by spasmodic tonic contractions of voluntary muscles All the common types of livestock are susceptible except poultry, which are relatively resistant Hagan and Bruner (1957) state that it requires about 350,000 times more toxin per gram of body weight to kill a chicken than is needed to kill a horse A list of the more highly susceptible animals would include man, horses, asses, mules, sheep, and goats Swine are more obviously susceptible when young, since a high percentage of cases occur at an early age, however, this may be at least partly due to the greater opportunities for infection at that time The common laboratory animals also are susceptible The distribution of the disease is essentially world wide, but it occurs most commonly in old farming areas with large livestock populations and heavily manured land

ETIOLOGY

Tetanus is caused by the toxins of *Clostridium tetani* (*Bacillus tetani*, Nicolaier's bacillus) The vegetative form of the organism is a slender rod, 0 4–0 6μ wide and 2–8μ long In young culture it is motile by means of peritrichous flagella and is Gram positive With age it stains erratically, usually becoming Gram negative It may occur singly in short chains or in filaments Spores are usually terminal in location and 2 to 3 times the diameter of the vegetative rod giving the sporulated rod the appearance of a spoon or drumstick It grows well on artificial media under fairly good conditions of anaerobiosis, particularly if glucose is added for enrichment Gelatin is liquefied and blackened Deep agar colonies are fluffy and spherical None of the common carbohydrates is fermented (Hagan and Bruner, 1957, Kelser and Schoening, 1918, Merchant and Packer, 1956)

Cl tetani and the disease which it produces are of historical interest The disease has been known and greatly feared for centuries As early as the fourth century B C. physicians believed tetanus was caused by the wind (Yu, 1930) In 1884, Carle and Rattone produced tetanus in a rabbit using material from a person who had died from 'lockjaw ' The same year, Nicolaier produced the disease in rabbits, guinea pigs, and mice by inoculating them with garden soil In 1889 Kitasato, using anaerobic methods, isolated the organism in pure culture and reproduced the disease in susceptible animals with the pure culture The following year (1890), von Behring and Kitasato published information regarding the formation of toxins by *Cl tetani*, showed that rabbits could be

PATHOLOGICAL CHANGES

Incision of the affected parts reveals an abundance of amber to blood tinged and watery to gelatinoid fluid While muscle tissue may be affected, as evidenced by dark red discoloration and edema, the edema fluid is more plentiful in the loose connective tissues such as the subcutaneous and intermuscular connective tissues Emphysema is generally mild Petechial or ecchymotic hemorrhages may be found in the serous membranes, and blood tinged serous fluid is often found in excess in the serous cavities Lymph nodes contiguous to the infected area are markedly swollen, hemorrhagic, and edematous, however, the spleen usually shows little change Heart, liver, and kidneys may show albuminous degeneration and acute general venous congestion may occur as a manifestation of heart failure from intoxication The blood is often dark and rather poorly clotted, particularly when passive congestion is marked The primary lesion is found at the point of infection Thus the location possibilities are numerous However, in the author's experience, malignant edema of swine has almost always been associated with the hog cholera immunization procedure (see below)

DIAGNOSIS

Malignant edema can generally be differentiated from anthrax in swine on the basis of history and lesions Anthrax occurs independently of vaccination procedures, and the lesions usually localize in the peri pharyngeal area, bringing about asphyxia through laryngeal edema It also shows almost no muscle involvement and no gas formation Although the gas gangrene type of wound infection in swine is usually due to *Cl septicum* or *Cl perfringens*, blackleg can occur and may be confused with it As indicated in the discussion of blackleg, and to further confuse the picture, infections with two or more organisms of this group may occur simultaneously Therefore, isolation and identification of the causative

organism (s) constitute the only reliable diagnostic means Even then there is room for error, since *Cl chauvoei* may escape detection in dual infections due to the masking or overgrowing effects of a less fastidious and more rapidly growing Clostridium Probably the most practicable means of identifying the organisms in culture or body fluids is by means of serum protection tests in susceptible laboratory animals However, it has been observed by Dafaala and Soltys (1951) that *Cl septicum* agglutinates the erythrocytes of various species of animals in a manner similar to certain filtrable viruses This hemagglutination can be neutralized by specific antisera Thus the technique might, under some conditions, find use in identification procedures

TREATMENT

Probably the most effective treatment of malignant edema is by large doses of penicillin and specific immune serum Since there is insufficient time to identify the organism (s) before instituting treatment and in view of the cost of antisera, penicillin alone is probably the most practical treatment In treatment of experimental clostridial infections of mice, the broad spectrum' antibiotics, Terramycin and Aureomycin, also have exerted favorable effect against *Cl septicum* (Taylor and Novak 1952) The greater value of local over systemic treatment was emphasized in this report, however, more information is needed regarding treatment of swine

IMMUNITY

Antisera and antitoxins can be produced for use against *Cl septicum*, but they find their chief use in the laboratory Formalized *Cl septicum* bacterin, especially in combination with *Cl chauvoei*, is the most practicable immunizing agent for veterinary practice, even so, its use is seldom justified in swine Numerous products, including toxoids, bacterins, and antitoxins are available for use in combating *Cl perfringens* infections in animals, but their use, likewise, is seldom justified in swine.

FIG. 19.1 — Idiopathic tetanus in pigs one week of age; infection may have occurred post-natally via the navel. Note "saw-horse" attitude caused by tetany of the strong extensor muscles. (Photo courtesy Department of Veterinary Pathology and Hygiene, University of Illinois.)
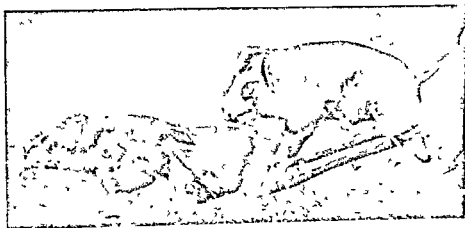
washed spores of the organism, when injected into healthy tissue, do not germinate, presumably because the oxygen tension is too high in such tissue. However, if a culture of the organism, which also contains toxin, is injected, germination occurs and the disease results. Likewise, it has been shown that the disease results with regularity when washed spores are introduced along with calcium chloride solutions (Bullock and Cramer, 1919). Thus it appears that tetanus spores germinate only in dead or injured tissues or those which are altered in such a way as to result in lowered oxygen tension. In addition, it has been observed that tetanus toxin so affects the leukocytes as to interfere with positive chemotaxis and phagocytosis of the spores (Delaunay, 1943).

The first symptom is a mild stiffness of muscles which may be local but is more often general. The progress of the disease is generally rapid enough, however, that the signs are distinctive within 24–36 hours of their initial appearance. The characteristic manifestation, from which the disease is named, is that of tonic or tetanic spasms of skeletal muscles. Although all such muscles are affected, the stronger ones overcome their weaker opposing muscles and produce the attitudes characteristic of the disease. Thus tetany of the muscles of the back, neck, and tail produces orthotonus and even opisthotonus, tetany of the muscles of the limbs produces a "saw-horse" attitude, the extensor muscles being stronger than the flexors (Fig 19.1), tetany of the muscles of mastication causes "lock-jaw." Difficulty in locomotion may be

manifested first in turning or backing, but the animal soon finds it difficult or impossible to walk or even stand (Fig 19.1). Pigs often show unusual erectness of the ears and some protrusion of the nictitating membrane (Fig 19.2). Spasms of the muscles of respiration permit only shallow and therefore rapid breathing. Tachycardia may be quite noticeable.

As the disease progresses, the animal becomes apprehensive and hypersensitive to sensory stimuli. Sudden movements, sharp noises, or a slap will cause or intensify the muscle spasms and protrusion of the nictitating membrane. In pigs the disease is almost always generalized and fatal. This is especially true of very young pigs, of which the writer has never seen one survive. Death undoubtedly is due to anoxia from interference with respiratory and cardiac functions.
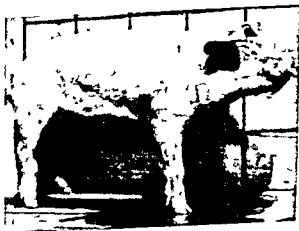
FIG. 19.2—Tetanus following castration. Note stiffness of legs in extension, erectness of ears, and protrusion of nictitating membrane. (Photo courtesy Department of Veterinary Pathology and Hygiene, University of Illinois.)

immunized against the toxin by the injection of small doses, and demonstrated that the blood serum from such rabbits could neutralize the toxin *in vitro* or *in vivo* Thus it was in connection with tetanus that (1) anaerobic techniques were first used, (2) bacterial toxins were first demonstrated, and (3) the foundation for serum therapy was laid

PATHOGENESIS AND CLINICAL SIGNS

Cl tetani gains entrance to the body via wounds The disease results most commonly from deep, penetrating, or puncture wounds those in which there is considerable tissue damage and those grossly contaminated with soil or manure Probably the most common portal of entrance in swine is via castration wounds Occasionally infection appears to occur via the unhealed navel shortly after birth, via the dental alveoli during eruption of teeth, or, possibly, through wounds caused by unclipped canine teeth or ' tusks ' There remains a small proportion of cases in which no likely portal of infection can be demonstrated these are often referred to as 'idiopathic tetanus Some such cases probably result from infection of small wounds which heal before tetany appears

Under favorable conditions the spores, having been carried into the wound, become vegetative, multiplying and forming toxin at the original site The toxin includes tetanolysin, a hemolytic portion, and tetanospasmin, the more important lethal fraction (Fleming, 1927) Tetanus toxin is one of the most poisonous substances known The organisms show little tendency to spread to other parts of the body However, the toxin, which is formed within the bacterial cell, apparently passes by diffusion out into the surrounding medium or environment and then spreads (Stone, 1951) to the central nervous system where it seems to produce its most dire results

There is no unanimity of opinion regarding the route by which the toxin reaches the brain Meyer and Ransom (1903) formulated the nerve transport theory, and Abel *et al* (1935) concluded that the toxin reached the brain only via the arterial blood after absorption from the site of local infection by blood and especially, lymph Both theories have had support, but the nerve transport theory appears to deserve and receive greater credence (Doerr and Seidenberg 1935 Zuger and Friedemann, 1938, Friedemann *et al*, 1939, 1941) Hogger (1939) noted phenomena which he said were explainable by neither of these theories He pointed to diffusion of toxin through the tissue spaces and an effect upon the muscles *through action on the nerve tissue*

Yokoi (1954) believes that the tetanus toxin not only becomes fixed in the central nervous system tissues, but is changed into some new toxic substance, differing from strychnine in this respect He believes that the reflex convulsions result from the effects *of the toxin* on the spinal cord, while rigidity of muscles at rest is under control of the brain Therefore, when tetanus toxin is injected directly into the brain or into the main cerebral arteries, the animal dies within a short time without showing typical tetany Baylis *et al* (1952) reported that transection of the spinal cord in the lumbar region prevents death in rabbits (such as occurs in controls) following injection of tetanus toxin into the sciatic nerve trunk It is inferred that transection of the spinal cord interrupts the pathway by which the toxin must reach the vital centers of the medulla oblongata to cause death Ponomarew (1928) concluded from investigations of tetanus in dogs that lymph in the nerve trunks has a definite uninterrupted centripetal flow which plays a part in carrying the toxin to the brain

The incubation period for tetanus is usually 1 to 3 weeks but occasionally is shorter or much longer The length of the incubation period is probably influenced not only by the numbers of infecting organisms but by the conditions set up in the contaminated wound and particularly by the amount of tissue damage incurred It has been shown, for example, that

precipitated toxoid will produce appreci able immunity, but 3 doses about 3 weeks apart provide excellent protection for a year or more, which is longer than most market hogs are kept Breeding stock can be given a "booster" injection in the event of a dangerous wound

EPIZOOTIOLOGY AND CONTROL

The spores of *Cl tetani* are extremely resistant, and there are no practical means of eliminating them from the environment Losses from tetanus, though generally sporadic, may be enzootic due to heavy concentrations of spores in the soil and inadequate precautions against the disease Kaplan (1943) reported, for example death of about 60 pigs in two outbreaks constituting losses of about 10 per cent

There remain three points at which attempts can be made to break the cycle of disease First, since it is a wound infection disease, every effort should be made to prevent the incurring of unnecessary wounds by removing from the environment any sharp objects such as bare nail points projecting from boards and by clipping the sharp points from the tusks or 'needle teeth of newborn pigs Secondly, a clean

environment should be provided so far as possible e g farrowing pens should be periodically sanitized and kept as free of manure as possible, umbilical cords should be tied off and treated with antiseptic soon after birth, and pigs should be turned out on clean pasture — not into mud holes — after castration The castration operation should be performed with every rea sonable precaution to asepsis and establish ment of good wound drainage If all these precautions are duly observed tetanus is not likely to follow Thirdly, if all these precautions do fail or if not all can be properly carried out there remains the possibility of immunization In fact still another prophylactic measure has been suggested by Novak *et al* (1949) Work ing with mice, they demonstrated consid erable prophylactic value from administer ing procaine penicillin G in oil within 6 hours after infection Such injections were followed by maintenance of therapeutic blood levels for periods up to 4 days Their observations suggest that use of some of the still newer and possibly longer acting penicillin preparations may be of consid erable practical value in tetanus prophy laxis

REFERENCES

General

FIELD, H I, AND GIBSON E A 1955 Studies on piglet mortality 2 *Clostridium welchii* infec tion Vet Rec 67 3

HAGAN, W A, AND BRUNER D W 1957 The Infectious Diseases of Domestic Animals, 3rd ed Comstock Publ Assoc, Ithaca, N Y

KELSER, R A, AND SCHOENING H W 1948 Manual of Veterinary Bacteriology 5th ed Williams and Wilkins Co, Baltimore, Md

MERCHANT, I A, AND PACKER, R A 1956 Veterinary Bacteriology and Virology, 4th ed Iowa State University Press Ames Iowa

RECORDS, E, AND HUBER, M 1931 *B histolyticus* infection in a hog Jour Amer Vet Med Assn 78 863

Blackleg

AITKEN, W A 1949 Penicillin in blackleg No Amer Vet 50 111

ARLOING, S, CORNEVIN CH, AND THOMAS 1887 Le Charbon symptomatique a laveul and ed Asselin et Co, Paris

ECCLESTON, E L 1950 Blackleg in swine Vet Med 45 253

FRANKLIN, O M, AND HASLAM T P 1946 The strength and comparison of blackleg vaccines Jour Infect D s 19 103

GLASSER, K, HUPKA, E AND WETZEL, R 1950 Die krankheiten des Schweines 5th ed M u H Schaper Hannover

CUELENÈRE, G 1935 L'inference du "Clint chauffeur" sur le taureau Arch Vet Ital 6 3

HAGAN, W L, AND BRUNER D W 1957 The Infectious Diseases of Domestic Animals Comstock Publ Assoc Ithaca N Y

PATHOLOGICAL CHANGES

Rigor mortis appears early in the animal dead from tetanus, however, there are no significant gross lesions characteristic of tetanus The blood is usually dark red and may be poorly clotted Pulmonary congestion and edema may be noticeable There may also be a few petechial or ecchymotic hemorrhages in the serous membranes In humans, Baker (1943) has reported histopathological evidence of irreversible damage to motor nuclei of certain cranial nerves which may explain death from respiratory and/or cardiac failure However, similar studies in swine apparently have not been reported

DIAGNOSIS

Despite the fact that there are no characteristic gross lesions in tetanus, the diagnosis usually presents no particular difficulties The symptoms in swine are not likely to be confused with those of any other disease with the exception of strychnine poisoning which has seldom if ever been reported and is not likely to occur Symptoms of tetanus are often accompanied by a history of recent castration or the presence of a contaminated or infected wound Therefore, clinical diagnosis is based upon history and symptoms It is confirmed at necropsy by the absence of significant gross lesions and, in the event the infected wound can be demonstrated, the presence of the characteristic 'drumstick' bacterial forms in it It is seldom necessary to go beyond the clinical examination to establish the diagnosis beyond reasonable doubt

TREATMENT

There is little evidence that swine can be successfully treated, once symptoms of tetanus have appeared Apparently once the toxin has become 'fixed' in the central nervous system, it cannot be altered by treatment. Some benefit has been reported in connection with other species from certain treatments which will be mentioned briefly here for the sake of academic interest and possible experimental use in swine

Sedatives may be used for palliation but probably have no further value Aureomycin, Terramycin, penicillin, and possibly, Chloromycetin reduce mortality in mice (Taylor and Novak, 1952) Cortisone, given orally, gives beneficial results in man (Lewis et al, 1954) There may be some connection between this fact and the report by Giroud et al (1941) that the hormone content of the adrenal cortex diminishes markedly in animals poisoned with either tetanus or diphtheria toxin The value of antitoxin appears to be limited to neutralization of toxin before it becomes fixed in the nervous system However, flooding the circulation with antitoxin as early as possible after symptoms appear may serve to neutralize any circulating toxin and prevent further absorption from the wound Injection of antitoxin around the wound may also assist in localizing the effects of the toxin

IMMUNITY

Swine are relatively susceptible to tetanus, their blood contains no demonstrable antitoxin Exposure should be expected to result in disease, therefore, unless prophylactic measures are taken If exposure cannot be avoided with reasonable assurance, immunization procedures are in order Antiserum is effective if present in the system in sufficient concentration when needed However, under most circumstances the period of protection desired is greater than a single injection of antiserum can be relied upon to provide Effective and lasting active immunity can be engendered by the use of the toxoid or anatoxin Lowenstein (1921) reported that tetanus toxin, treated with formol and exposed to diffuse light for some time, lost its toxicity but retained its antigenicity The effectiveness of toxoid (formalized toxin) was shown in guinea pigs by Fortner in 1928 since that time an improved product has been obtained by alum precipitation A single injection of the alum

FLEMING, W L 1927 Studies on the oxidation and reduction of immunological substances VII
    The differentiation of tetanolysin and tetanospasmin Jour Exper Med 46 279
FORTNER, J 1928 Immunization with formalinized tetanus toxin Zeitschr f Hyg u Infek
    tionskr 108 773
FRIEDEMANN, U , HOLLANDER, A , AND TARLOV, I M 1941 Investigation on the pathogenesis of
    tetanus Jour Bact 41 75
———, ZUGER, B , AND HOLLANDER, A 1939 Investigations on the pathogenesis of tetanus II
    The influence of section of the nerve on the neutralizing of intramuscularly injected
    tetanal toxin by circulating antitoxin Jour Immunol 36 485
GIROUD, A , GIROUD, P , AND MARTINET, M 1941 Action of toxins on corticoadrenal function C
    R. Soc. Biol (Paris) 135 565
HAGAN, W A , AND BRUNER, D W 1957 The Infectious Diseases of Domestic Animals 3rd ed
    Comstock Publ Assoc, Ithaca N Y
HOGGER, D 1939 Uber die Ausbreitung und den Angriffspunkt des Tetanustoxins im Fallen
    des localen Tetanus Zeitschr f Hyg u Infektionskr 121 663
KAPLAN, M M 1943 An unusual epizootic of tetanus in young pigs Middlesex Vet 3 8
KELSER, R A , AND SCHOENING H W 1918 Manual of Veterinary Bacteriology 5th ed Williams
    and Wilkins Co , Baltimore, Md
KITASATO, S 1889 Ueber den Tetanusbacillus Zeitschr f Hyg 7 225
LEWIS, R. A , SATOSKAR, R S , JOAG, G G , DAVE, B T., AND PATEL, J C 1954 Cortisone and
    hydrocortisone given parenterally and orally in severe tetanus Jour Amer Med Assn
    156 479
LOWENSTEIN, E 1921 Immunization with non toxic toxins Deutsche med Wschr 47 833
MERCHANT, I A , AND PACKER, R A 1956 Veterinary Bacteriology and Virology, 5th ed Iowa
    State College Press, Ames Iowa
MEYER, H , AND RANSOM, F 1903 Untersuchungen uber den Tetanus Arch f Exper Path u
    Pharm 49 369
NICOLAIER, A 1884 Ueber infectiosen Tetanus Deutsche med Wschr 10 842
NOVAK, M , GOLDIN, N , AND TAYLOR W I 1949 Tetanus prophylaxis with penicillin procaine
    G Proc Soc. Exper Biol and Med 70 573
PONOMAREW, A. W 1928 Zur Frage der Pathogenese des Tetanus und des Fortbewegungs
    mechanismus des Tetanustoxins langs dem Nerven Zeitschr gesam Exper Med 61 93
STONE, J L 1954 On the mode of release of tetanus toxin from the bacterial cell Jour Bact
    67 110
TAYLOR, W I , AND NOVAK, M 1952 Antibiotic prophylaxis of experimental clostridial infec
    tions I Antibiotic prophylaxis of tetanus Antibiot and Chemother 2 517
YOKOI, W 1954 Experimental studies on tetanus convulsions Nagoya Med Jour 2 87
YU, H 1930 Tetanus in ancient Chinese medical literature Nat Med Jour China 16 297
ZUGER, B , AND FRIEDEMANN, U 1938 Investigations on the pathogenesis of tetanus Proc Soc.
    Exper Biol and Med 38 283

HELLER H H 1920 Etiology of acute gangrenous infections of animals a discussion of black leg braxy malignant edema and whale septicemia Jour Infect Dis 27 385

HUTYRA F MAREK J AND MANNINGER R 1949 Special Pathology and Therapeutics of the Diseases of Domestic Animals 5th Engl ed Alexander Eger Chicago

KELSER R A AND SCHOENING H W 1948 Manual of Veterinary Bacteriology 5th ed Williams and Wilkins Co Baltimore Md

KITT TH 1888 Ueber Abschwachung des Rauschbrandvirus durch stromende Wasserdampfe Centralbl f Bakt 3 5/2

KOLLE W AND WASSERMANN A V 1928 Handbuch der pathogenen Mikroorganismen 3rd ed G Fischer Jena Vol 4 p 1217

LECLAINCHE E AND VALLEE H 1925 Sur la vaccination contre le charbon symptomatique C R Soc Biol (Paris) 92 1273

MERCHANT I A AND PACKER R A 1956 Veterinary Bacteriology and Virology 5th ed Iowa State College Press Ames Iowa

MEYER K F 1915 The etiology of symptomatic anthrax in swine Jour Infect Dis 17 458

NITTA N 1918 Investigations on blackleg immunization Jour Amer Vet Med Assn 53 466

ORFEUR N B AND HEBELER H F 1953 Blackquarter in pigs Vet Rec 65 822

PERCIVAL R C LEAMING J D MARTINI F V AND TONELLI G 1953 Aureomycin in the treatment of experimental *Clostridium chauvoei* infection in sheep Cornell Vet 43 92

SCHOBL O 1910 Ueber die Aggressinimmunisierung gegen Rauschbrand Centralbl f Bakt Abt I Orig 56 395

STERNE M AND EDWARDS J B 1955 Blackleg in pigs caused by *Clostridium chauvoei* Vet Rec 67 314

Malignant Edema

DAFAALA E N AND SOLTYS M A 1951 Studies on agglutination of red cells by clostridia I *Cl septique* Brit Jour Exper Path 32 510

ERMENGEN E VAN 1896 Untersuchungen uber Falle von Fleischvergiftung mit Symptomen von Botulismus Centralbl f Bakt Abt I 19 442

GEIGER W 1929 Gasoedeme be m Schwein Deutsch tierarztl Wschr 37 561

HAGAN W A AND BRUNER D W 1957 The Infectious D seases of Domestic Animals 3rd ed Comstock Publ Assoc Ithaca N Y

KELLAWAY C H REID G AND TRETHWIE E R 1941 Circulatory and other effects of toxin of *Cl septique* Australian Jour Exper Biol and Med Sci 19 297

KELSER R A AND SCHOENING H W 1948 Manual of Veterinary Bacteriology 5th ed Williams and Wilkins Co Baltimore Md

MCLEAN D AND ROGERS H J 1943 Early d agnosis of wound infection with special reference to mixed infections Lancet 244 707

MERCHANT I A AND PACKER R A 1956 Veterinary Bacteriology and Virology 5th ed Iowa State College Press Ames Iowa

MULLALLY G T 1941 Anaerob c infections a1d gas gangrene Lancet 240 269

REED G B ORR J A AND SMITH D 1941 Fibrinolytic action of gas gangrene anaerobes Proc Soc Exper Biol and Med 47 228

TAYLOR W I AND NOVAK M 1952 Antib otic prophylaxis of experimental clostridial infections Antibiot and Chemother 2 639

VARNELL J 1919 Contribution to the experimental study of gaseous gangrene C R Soc B ol (Paris) 82 493

WARRACK G H BIDWELL E AND OAKLEY C L 1951 The beta toxin (desoxyribonuclease) of *Cl septicum* Jour Path and Bact 63 293

Tetanus

ABEL J J HAMPIL B AND JONAS A F JR 1935 Researches on tetanus 3 Further experiments to prove that tetanus toxin is not carried in peripheral nerves to the central nervous system Bull Johns Hopkins Hosp 56 317

BAKER A B 1943 Medullary involvement in tetanus Amer Jour Path 19 709

BAYLIS J H JOSEPH J MACKINTOSH J MORGAN R S AND WRIGHT P 1952 The effect of transect on on the ascent of tetanus toxin in the rabb ts spinal cord Jour Path and Bact 64 47

BEHRING VON E AND KITASATO S 1890 Ueber das Zustandekommen der Diphtherie Immunitat u1 d der Tetar us Immunitat bei Thieren Deutsche med Wschr 16 1113

BULLOCK W E AND CRAMER W 1919 A new factor in the mechanism of bacterial infection Proc Roy Soc London 90B 513

CARLE AND RATTONE 1884 Studio esperimentale sull e ologia del Tetano Giorn d R Accad di Med di Torino (3) 174

DELAUNAY A 1943 Recherches sur la phagocytose V Chimiotactisme leucocytaire et toxines microbiennes Rev Immunol (Paris) 8 30

DOERR R AND SEIDENBERG S 1935 Dynamische Aktivierung des toxisch induzierten lokalen Tetanus Zeitschr f Hyg u Infektionskr 117 561

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CHAPTER 20

Dysentery

Swine dysentery is an infectious, readily transmissible disease It is also known as bloody diarrhea, bloody scours, bloody flux, and black scours It is not known to be transmissible to any species but swine The disease was first reported by Whiting, Doyle, and Spray in 1921 It is apparently rather widespread throughout the world, wherever hogs are raised

Reports from Italy, Spain, Hungary, Switzerland, Holland, and Australia indicate that the disease has been observed in those countries Swine of all ages are susceptible, but the death rate is usually higher in young swine than in older hogs

ETIOLOGY

The specific cause of swine dysentery is present in the intestine, particularly in the large intestine, and is given off in the feces Its presence in the body of an infected animal appears to be limited largely to the cecum, colon, and rectum The most likely cause is a vibrio Several different vibrios may be found in the gastrointestinal tracts of apparently healthy hogs Most of these are probably harmless The vibrio which appears to be etiologically significant has growth requirements and cultural characteristics similar to those of *Vibrio fetus* When first isolated, it grows best in an atmosphere containing about 15 per cent of carbon dioxide or in sealed tubes or culture dishes with small air spaces At first the growth is scant, consisting of small,

discrete, translucent colonies Tryptose agar, plus about 10 per cent of blood is a suitable culture medium for primary isolation The vibrio is actively motile and Gram negative, and causes little or no change in the usual culture media Cultures usually die out within 4 to 6 days at incubator temperature, but may remain alive for several weeks when frozen Vibrio coli has been suggested as a name for this microorganism

Salmonella choleraesuis is frequently found in hogs that have dysentery It is generally known that cultures of *Sal choleraesuis* can cause enteritis However, the disease thus caused is not identical with swine dysentery The symptoms and lesions are different, and the disease caused by Salmonella cultures does not spread, on pen contact, to the extent that dysentery does

CLINICAL SIGNS

The clinical signs of swine dysentery are diarrhea, dehydration, and loss of weight The incubation period is usually from 1 to 2 weeks However, the disease may not appear in contact animals for several weeks or months after a carrier animal is brought into the herd One of the early symptoms often noted is the sunken or hollow appearance of the flanks In a severe attack, the sunken flank may be conspicuous within 24 hours after the onset of the disease There is usually not much rise of body

cholera there may be various quantities of blood in the large intestine without much mucus Deep necrosis of the intestinal wall often occurs in hog cholera of several days duration In typhlitis and colitis due to other causes such as Salmonella there may be varying degrees of necrosis and slough ing but blood and mucus are usually not conspicuous The occasional cases of mu coid enteritis of unknown etiology can usually be distinguished from dysentery by the absence of blood in the large intestine and in the feces Thus far serologic tests have been of little or no diagnostic value

TREATMENT

There are some treatments which re duce the economic loss from swine dysen tery by alleviating symptoms and decreas ing mortality It is doubtful if any avail able treatment definitely cures the disease Recurrences are to be expected with any treatment now available Arsenic and some antibiotics are generally used as treatments Any one of these treatments usually al leviates symptoms and reduces death losses The preparation of arsenic generally used is sodium arsanilate but other arsenic compounds are also used The dose of ar senic should be carefully adjusted so as to avoid the danger of poisoning

Arsenic compounds are commonly given in doses sufficient to supply 0 8 gram of elemental arsenic per day per animal However, as the organic compounds are generally used the amount of arsenic given is often somewhat greater than 0 8 gram per day Sodium arsanilate is given either in the feed or drinking water Four ounces are mixed with one ton of feed One pound is dissolved in 800 to 1 000 gallons of drinking water Special care should be used to make certain that the arsenic com pound is thoroughly mixed with the feed or water Treatment is continued for 3 to 5 days and repeated later if necessary It should be stopped at least a week be fore slaughter

Arsenic preparations should always be handled cautiously Children should never be allowed to have access to arsenic All farm animals other than hogs being treated should be kept away from the medicated feed or water Contamination of food or containers and contact with the eyes or the exterior of the human body should be carefully avoided

Some antibiotics have been used as treatments and have been as effective as anything available Streptomycin and baci tracin have been tried rather extensively No doubt other antibiotics will prove to be as effective It is often more advantageous to give an antibiotic in the drinking water than in the feed In such cases a water soluble product is preferable The dose of streptomycin that has been used quite generally is 1 gm per pig per day Daily treatments are given for 3 to 5 days and repeated later if necessary The dose of bacitracin which has been reported is 100 000 units for 6 days

IMMUNITY

It is difficult to determine the degree of immunity which results from an attack of swine dysentery because of the tendency of the disease to recur after remissions of variable length

Some clinically recovered swine remain free from symptoms for several months. It is impossible to know what portion of these apparently recovered animals remain car riers and possible spreaders of infection Whatever the acquired immunity it has not been of much practical value in con trolling the disease Effective artificial means of producing immunity have not been developed. Remissions in dysentery such as also occur in many other diseases, often give rise to the erroneous impression that the disease has stopped

EPIZOOTIOLOGY AND CONTROL

Swine dysentery is usually spread by moving hogs from infected herds to healthy ones. Many exposed animals are infected during the incubation period of the dis ease, hence symptoms may appear within a few days So an animals which have gone through an outbreak remain carriers al though they may appear to be healthy

temperature Frequently the appetite is affected very little Animals which scour severely may continue to eat However some affected animals show signs of marked illness Occasionally the finding of a dead hog which was not seen to be sick is the first evidence of the disease The morbidity rate varies from a few animals to nearly 100 per cent depending largely on the closeness of contact between animals in the herd The mortality in untreated herds varies from a few animals to more than 50 per cent The average death loss is about 25 per cent of an infected herd if nothing is done to stop the disease

The diarrhea of swine dysentery may not have any distinguishing characteristic during the first day By the second or third day the bowel discharge contains mucus and/or blood The relative amounts of blood and mucus vary but both are usually present In young swine the blood is easily recognized In older animals the blood may be changed so as to give a dark color to the feces hence the name black scours As the disease progresses the bowel d scharge may contain less blood and there may be present considerable quantities of a grayish granular or flaky material The affected animals show sunken flanks within 2 or 3 days after the onset of the disease and may lose considerable weight during the following week or two The course of the disease may vary from a few days to 3 or 4 weeks or longer Most of the deaths occur within the first 2 weeks after symptoms appear There are nearly always recurrences at varying intervals In a good many cases there are recurrences at intervals of about 30 days Some animals die during a recurrence

PATHOLOGICAL CHANGES

The characteristic lesions are found in the large bowel The stomach mucosa of ten shows hyperemia and sometimes hemorrhages Since gastritis occurs in several swine diseases it has little specific diagnostic significance The small intestine usually appears normal The sharp limitation of intestinal lesions to the large intestine is

characteristic of dysentery Some degenerative changes may occur in the liver Nephrosis may also be seen

Sometimes marked reddening of the large intestine is readily apparent when the abdomen is opened In an early stage of the disease the mucosa of the large intestine may show small discrete hemorrhages as well as diffuse congestion The colon content consists of blood and mucus mixed with the feces The characteristic colon content is most easily seen in the dependent portions of the colon The presence of mucus together with blood helps to distinguish dysentery from some other diseases particularly cholera which occasionally causes copious hemorrhage in the large intestine

In a later stage of the disease the colic and cecal mucosa shows more or less superficial sloughing of diphtheritic exudate and surface epithelium This sloughed material in the form of granules or flakes becomes mixed with the intestinal content giving it a rice water appearance In this stage there may not be much blood in the colon but mucus is likely to be present Any necrosis that occurs is superficial Deep necrosis of the cecal or colic wall such as sometimes occurs in hog cholera and in colitis due to other causes rarely happens in dysentery

DIAGNOSIS

The clinical diagnosis of swine dysentery is usually easy It is based upon finding the bloody mucous bowel discharges from swine, some of which otherwise appear normal The bloody mucous feces may be found in the hog lot Consequently, an examination of the lots or pens where affected animals are kept may reveal the characteristic bowel discharges and thus aid greatly in the diagnosis The pathologic diagnosis in an early stage depends upon finding the injected hemorrhagic mucosa together with blood and mucus in the large intestine Typically these changes are limited to the large intestine In later stages there is diphtheritis with superficial necrosis and more or less sloughing In hog

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Salmonellosis

HISTORY

Salmon and Smith (1886a 1886b 1887 1889) isolated *Salmonella choleraesuis* from cases of hog cholera By means of cultures of this organism they produced a disease condition which resembled hog cholera in some respects and on the basis of this work *Salmonella choleraesuis* (Bacterium of hog cholera) for many years was considered to be the cause of hog cholera

This concept was disproved by de Schweinitz and Dorset (1903) Previously these workers had noted variations in the clinical manifestations and pathological findings among some of the outbreaks which prompted them to investigate the cause of those disease occurrences which were not associated with the hog cholera and the swine plague bacteria During the course of this work de Schweinitz and Dorset (1903) produced a disease in swine by subcutaneous inoculation of filtered body fluids which did not contain bacteria These filtrates in the absence of bacteria did not produce the disease in rabbits or guinea pigs This work constitutes the first etiological evidence of the disease now called hog cholera and which is caused solely by a filtrable virus In a later publication Dorset et al (1905) submitted additional data and proof conclusively establishing a filtrable virus as the cause of hog cholera without the introduction of *Salmonella choleraesuis*

The role of *Salmonella choleraesuis* as a primary pathogen is ably discussed by Dorset et al (1905) They state we do not deny the possibility of independent disease being caused by *B choleraesuis* In fact it is difficult to avoid a belief in such a possibility on account of the very considerable pathogenic power for hogs exhibited by many cultures of that organism when fed or administered intravenously Some later investigators have failed to consider this and other evidence in their work and interpretation Dorset et al (1905) also state that the conceivably lowered resistance caused by the virus permits the *Salmonella choleraesuis* to invade and if the filtrable virus is capable of lowering the resistance of hogs it would also be possible for other factors to have the same effect on resistance In view of the frequent isolations of *Salmonella choleraesuis* from hogs that were experimentally infected with filtered virus blood (containing no *Salmonella*) Dorset and co-workers point out that this agent may be a normal inhabitant in the bodies of apparently healthy hogs and when body resistance is lowered, bacterial invasion occurs and disease processes are started *Salmonella choleraesuis var kunzendorf* is the predominating type of *Salmonella* isolated

[317]

When such carriers introduce the infection into a herd, symptoms may not appear in the contact animals until the end of several weeks or months Infection may also occur as the result of healthy swine coming in contact with infected trucks, cars, stock yards, and barns Manure spreaders, tractors or other farm machinery or equipment may carry the infection from one farm to another Instances have occurred where infection was apparently carried a short distance by streams The only sure way to get rid of swine dysentery is to dispose of the entire infected herd and restock from a healthy source The causative agent probably does not survive long outside the hog under ordinary conditions, however, under special conditions such as freezing, the survival period may be considerably longer Perhaps there are other conditions under which the survival period may be considerably lengthened Consequently, the quarters and equipment used by infected swine should be thoroughly cleaned and disinfected after all the infected and exposed animals have been removed

REFERENCES

BALLMOOS P VON 1950 Über die Vibrionen Dysenterie des Schweines Schweiz Arch f Tier heilk 92 154

BOLEY, L E WOODS G T, HATCH, R D, AND GRAHAM, R 1951 Experimental therapy with sulfathaladine, sulfamethazine sodium arsanilate, and bacitracin in a natural outbreak of swine dysentery Cornell Vet 41 231

BRYANT, J B 1939 Swine enteritis in veterinary practice Proc 43rd Ann Meet U S Livestock San Assn

CARPENTER, L E AND LARSON, N L 1952 Swine dysentery treatment with 4 nitro and 3 nitro 4 hydroxy phenyl arsonic acids and antibiotics Jour Anim Sci 11 282

COLE, C R 1949 Vibrionic dysentery of swine 18th Ann Conf for Vet Ohio State Univ

DOYLE, L P 1939 Infectious types of swine enteritis Proc 43rd Ann Meet U S Livestock San Assn

——— 1940 The enteritis syndrome of swine No Amer Vet 21 213

——— 1944 A vibrio associated with swine dysentery Amer Jour Vet Res 5 3

——— 1945a Enteritis in swine Cornell Vet 35 103

——— 1945b Swine dysentery Jour Amer Vet Med Assn 106 26

——— 1948 The etiology of swine dysentery Amer Jour Vet Res 9 50

GRAY, L A 1939 Enteritis in swine No Amer Vet 20 27

HINDMARSH W L STEWARD F D AND HART, L 1940 Swine dysentery and Salmonella Australian Vet Jour 15 27

JAMES, H D AND DOYLE, L P 1947 Further studies with a vibrio as the etiologic agent of swine dysentery Jour Amer Vet Med Assn 111 47

McNUTT, S H, AND DACORSO, P 1947 Types of swine enteritis Proc 51st Ann Meet U S Live stock San Assn

MURPHY, J H 1941 Swine dysentery Vet Med 36 100

ROBERTS, D S 1956 Vibrionic dysentery in swine The isolation of a vibrio from an outbreak in New South Wales Australian Vet Jour 32 27

——— 1956 Studies on vibrionic dysentery in swine Australian Vet Jour 32 114

SALISBURY, J D SMITH, C R, AND DOYLE, L P 1951 Antibiotic treatment of swine dysentery Jour Amer Vet Med Assn 118 176

SCHMID G 1950 Mitteilung uber die Vibrionen Dysenterie des Schweines Schweiz Zeitschr Path und Bact 12 501

SCHENNIKOV, S T 1916 Anaerobic dysentery of pigs Veterinariza 15 20 (Abs No Amer Vet 28 436 1947)

STEENERSON, T L 1941 A practitioner s experience with infectious hemorrhagic dysentery of swine Jour Amer Vet Med Assn 99 210

TRUAX, E R 1941 Swine practice Vet Med 36 206

WHITING, R A DOYLE, L P AND STRAY, R S 1921 Swine dysentery Purdue Univ Agr Exp Sta Bull 257

WILSON, F M 1940 Enteritis in swine Jour Amer Vet Med Assn 96 141

frequently in young animals Attention is called to the carrier state in animals without visible symptoms

From 1,056 outbreaks of salmonellosis in swine 810 of the isolations were *S cholerae suis* (Table 21 1) These results were influenced to some extent by the inclusion of isolations from feces and intestinal contents of one large institutional group of swine held under experimental conditions (Bruner and Moran, 1949)

TABLE 21 1
SALMONELLA IN SWINE *

Salmonella in Swine	Number of Outbreaks	Salmonella in Swine	Number of Outbreaks
paratyphi B	7	*saintpaul*	2
sandiego	1	*derby*	20
californa	1	*typhi murium*	79
bredeney	21	*4, 5 12*	2
choleraesuis	810	*norwich*	1
montevideo	1	*thompson*	1
oranienburg	2	*bareilly*	4
muenchen (oregon)	11	*manhattan*	5
newport	13	*6, 8*	1
kentucky	1	*enteritidis*	7
panama	3	*pullorum*	2
anatum	15	*meleagridis*	2
give	10	*lexington*	1
newington	3	*newbrunswick*	4
ill nois	1	*senftenberg*	7
mississippi	1	*wichita*	1
worthington	11	*poona*	1
cerro	2	*urbana*	1

* From Bruner and Moran (1949)

Hormaeche and Salsamendi (1939) isolated 85 strains of Salmonella from the mesenteric lymph nodes of normal swine in Uruguay Only one strain of *S cholerae suis* was reported The other Salmonella strains consisted of

derby	25
newport	13
bredeney	4
chester	2
newington	1
muenchen	1
typhimurium	19
anatum	12
montevideo	4
paratyphi B	2
london	1

Rubin *et al* (1942) examined the lymph nodes of 40 groups of hogs, each consisting of 25 animals and 50 individual hogs, all of which were passed for food after veterinary inspection From 19 of the 40 groups and from 10 of the 50 individual hogs 212 strains of Salmonella were recovered, among which were 13 species *S cholerae suis* was isolated from four of the specimens Fournier *et al* (1953) isolated 35 strains of Salmonella from the lymph nodes of 360 hogs at the Saigon abattoir, 23 of the strains being *S choleraesuis* Galton *et al* (1954) studied the incidence of Salmonella in Florida as related to the origin of pork sausage which was contaminated with Salmonella when it reached the market. They show that the mixing and mingling of hogs held in the pens at the sales barns and in the holding pens of packing plants provide the means for exposure and spread of Salmonella from animal to animal Salmonella organisms were isolated from 12 of 16 samples taken from the drinking water of these pens Their observations indicate that *S choleraesuis* has a wide distribution among swine They conclude that the presence of the organism in the intestinal contents and in the lymph nodes of abattoir hogs must be interpreted in the light of the previous history of the animals Saphra and Wasserman (1954), reporting on 4,000 Salmonella infections in man, found 290 or 7 2 per cent caused by *S choleraesuis*, which took fifth place in the order of frequency The organism was highly pathogenic for man often causing septicemia

During a five year period, 1950–54, Bruner (1956) studied 731 cultures of Salmonella isolated from domestic animals in New York State which were submitted for antigenic analysis Twenty two of the isolations were *S choleraesuis*, all of which came from swine Other types originating from swine in that study include one *S typhimurium*, two *S enteritidis*, and one *S senftenberg* He also found that asymptomatic carriers of Salmonella are not uncommon

from swine in the United States (Bruner and Edwards, 1940).

DESCRIPTION

Synonyms   Bacterium of hog cholera, *Bacterium cholerae suis, Bacterium suipestifer, Salmonella suipestifer. Salmonella choleraesuis* is the type species of this genus (Breed *et al.*, 1948). It was named in honor of D E. Salmon, who with Theobald Smith described and associated this organism with the cause of hog cholera. It is a rod shaped organism, 0 6 to 0.7 by 3.0 μ (Fig. 21 1), Gram-negative, a facultative aerobe, usually motile, with 4 to 5 peritrichous flagella, it occurs singly. Optimum growth occurs at 37°C. Gelatin is not liquefied On agar the colonies are moist, translucent, and grayish (Fig. 21.2). Litmus milk develops slight acidity followed by definite alkalinity. Hydrogen sulfide is not produced by *Salmonella choleraesuis*, but variety *kunzendorf* is characterized by the production of hydrogen sulfide.

FERMENTATION REACTIONS

Acid and gas are produced from glucose, fructose, galactose, mannose, xylose, maltose, glycerol, mannitol, dulcitol, rhamnose, sorbitol, and dextrin. Lactose, sucrose, arabinose, inositol, salicin, inulin raffinose, and trehalose are not affected. The classification of Salmonella is based mainly on the antigenic structure and biochemical properties of the group which have been determined by Bruner and Edwards (1940). The organism is destroyed in one hour at 56°C., in 20 minutes at 58°C, and in 15 to 20 minutes by the common disinfectants.

DISTRIBUTION

Salmonella spp. have a worldwide distribution. Although the pig is considered the reservoir for *S. choleraesuis*, this species has been recovered also from man, cattle, dogs, chickens, turkeys, foxes, and canaries Twelve other types of Salmonella in addition to *S. choleraesuis* were isolated from hogs by Rubin and co-workers (1942).

Bruner and Moran (1949) report on the derivation and distribution of 2,788 Salmonella cultures from animals other than man or fowls. From 1,056 outbreaks in swine 2,119 cultures of Salmonella were isolated; this represents 76 per cent of the total isolations from animals in that series Their records confirm other observations that outbreaks of salmonellosis occur more
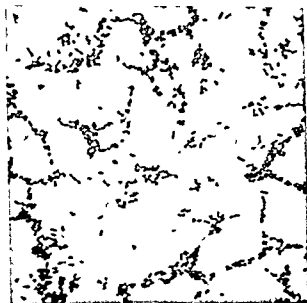
FIG. 21.1—Salmonella choleraesuis. X 920. (Biester, Iowa State College)

FIG. 21.2—Colonies of S. choleraesuis; a 48-hour culture. (Biester, Iowa State College.)

He isolated *S choleraesuis* from the lungs of 7 of the 13 pigs which had pneumonia

RELATION OF *S CHOLERAESUIS* TO ENTERITIS

In 1922 when laboratory and field studies were inaugurated in Iowa on a clinical entity in swine referred to as necrotic enteritis it soon became evident that more than one form of enteritis was present (Murray *et al*, 1927) From these clinical cases showing diarrhea and passing particles of croupous membrane pure cul tures of *S choleraesuis* were isolated Gross blood was not observed in the intestines or in the excreta of these cases Two out breaks of enteritis in this series differed from the others and from these *S cholerae suis* was not isolated Gross blood oozed through the intestinal lesions of these two cases They will be discussed briefly under differential diagnosis It was observed throughout the studies that cases of en

FIG 21 3—Balantidium coli in necrotic tissue of colon X 110 (Biester Iowa State University)

teritis from which *S choleraesuis* was iso lated invariably came from premises on which poor sanitation was practiced and poor rations were fed

Clinical Observations

The ages of animals in the field cases of enteritis studied varied from 3 to 6 months and the weights ranged from 18 to 110 pounds The experimentally pro duced subjects weighed from 25 to 70 pounds Temperatures ranged from sub normal to 107° F depending upon the stage of the disease Field cases presented rough staring hair coats and showed un thriftiness and sometimes extreme emacia tion The blood showed a slight decrease in hemoglobin and an increase of white cells especially the mononuclear cells The cases produced experimentally by feeding cultures of *S choleraesuis* showed higher hemoglobin readings and red cell counts during the few days after the culture was fed This was due to the rapid elimination of body fluids during diarrhea before sig nificant quantities of cellular elements were lost Similar hematologic changes are found in typhoid fever of man during the stages of sweating and diarrhea In severely infected animals considerable peritoneal exudate and a few petechiae on the serosa of the intestine and at times on the serosa of the stomach are present

Stomach The mucosa of the fundus usually was bright red and covered with a thin film of tenacious mucus Necrosis was sometimes present

Small intestine The changes increased in severity from the duodenum to the ileum In the ileum the lesions ranged in degree from advanced acute catarrhal ilei tis with marked edema and leukocytic in filtration to a more severe condition char acterized by diffuse cellular infiltration and by many distended crypts which bulged as a result of accumulations of exudate and inspissated contents A heavy layer of caseated material appeared above the surface un der which was found a zone of karyor

Numerous other reports on the occurrence and distribution of Salmonella in swine are reported from many countries Differences among species occurrence in various areas may be influenced by differences in husbandry practices and other factors The general conclusion is that Salmonella has a wide distribution and may be found in animals that show no visible signs of disease, but this does not preclude their role as primary pathogens under suitable conditions

INCIDENCE OF SALMONELLA IN HOG CHOLERA

The incidence and relation of Salmonella to hog cholera infection is evaluated in an excellent manner by Dorset and co-workers (1905) Ten Broeck (1918), during the course of experimental work isolated Salmonella from 16 per cent of hogs infected with hog cholera virus Uhlenhuth et al (1929) found S choleraesuis in 76 of 178 cases of hog cholera In a large herd where 150 pigs were exposed to hog cholera 104 succumbed and notwithstanding the presence of severe intestinal lesions in 78 cases S choleraesuis was isolated only 15 times They quote Preisz as finding Salmonella in 31 of 80 cases of hog cholera from different sources

Lutje (1938–39) in Germany collected data for a period of 15 years ending May 1, 1938 on the Enteritisbakterien in the pig The spleen, liver, and kidney of 7 778 pigs were examined bacteriologically From the carcasses of 552 animals with hog cholera he isolated Salmonella 41 times and from 245 slaughtered animals (presumably conservation slaughter of hog cholera hogs) Salmonella organisms were recovered from five From 3 131 swine which had died from diseases other than hog cholera 91 isolations of Salmonella alone were made and in 10 instances both Salmonella and Erysipelothrix were isolated from the same animal Among 3 850 slaughtered hogs which were free of hog cholera 32 isolations of S choleraesuis were

made, and in three cases both Salmonella and Erysipelothrix were obtained

PATHOGENICITY OF S CHOLERAESUIS

The role of S choleraesuis as a pathogen may be influenced by factors that lessen the resistance of the host Among these may be included other infections, notably hog cholera, inadequate nutrition, parasitoses exposure, and confinement where soil saturation is intensified with various forms of enteric parasites and bacteria including S choleraesuis The nature of the bacterial culture and the media on which it is grown may also influence the ability to produce experimentally enteritis or disease in the pigs The ability of the organism to produce enteritis in the pig is enhanced when it is grown on a medium containing serum or one similar to that originally devised by Huntoon (1918) to replace serum media The influence of bacteriophage also might influence the pathogenic behavior of S choleraesuis Dale et al (1944) found a bacteriophage in swine feces which destroyed S choleraesuis in vitro

In evaluating the pathogenic power of S choleraesuis, consideration should be given also to the fact that the pig by reason of its confinement environment and eating habits since domestication has acquired some resistance to enteric infections Balantidium coli of the pig is an example (Fig 21 3) Large masses of closely packed Bal coli were found in some cases of swine enteritis that were characterized by extensive necrosis of the intestine but only in rare instances did this protozoon aggressively invade beyond the zone of necrosis (Fig 21 3)

FORMS OF SALMONELLA INFECTIONS

S choleraesuis infections may appear in the form of septicemia pulmonary involvement, or as enteritis

McBryde (1937) studied bacteriologically 19 pigs showing enteritis These were obtained from a garbage feeding establishment that experienced considerable losses

FIG 21 5—Large colon
Note zones of caseation,
karyorrhexis, and leuko
cytosis X 27 (Biester, Iowa
State University)
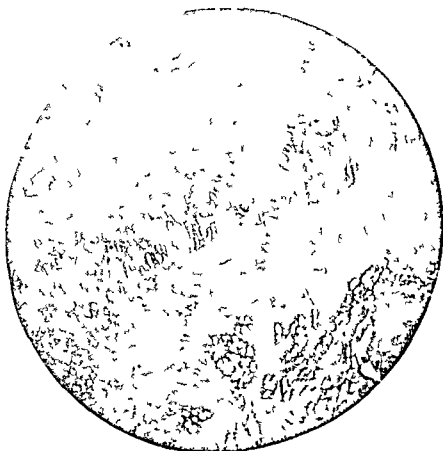
FIG 21 6—Cecum Enteri
tis induced by feeding
cultures of S choleraesuis
Note necrosis and infil
tration extending into
inner circular muscle of
cecum X 440 (Biester,
Iowa State University)
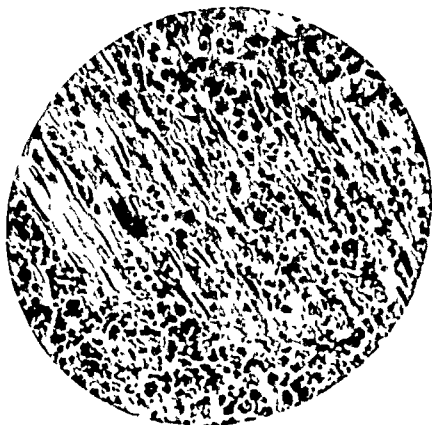
rhexis   Some agminated lymph nodules showed leukocytic infiltration and caseation necrosis

Cecum and large colon. Grossly the wall usually appeared two to four times normal thickness, the mucosa being covered by an extensive layer of necrotic tissue (Fig. 21.4). When this diffuse, leathery grayish-yellow covering, or diphtheric membrane, was scraped off, a denuded red granular surface was seen. In less advanced cases the membrane was focal and in some areas it could be pulled off. The advanced field cases often presented an intestinal wall that was described as resembling a garden hose in consistency.

Microscopically the mucosa often had undergone almost complete necrosis (Fig. 21 5). In the advanced stage the lesion consisted of three zones: (1) caseation, varying in thickness to 3 mm.; (2) karyorrhexis, frequently extending into the submucosa and in some cases involving the muscle layers (Fig. 21 6); (3) a variably dense zone of leukocytes appearing beneath

the karyorrhectic zone. In some instances the process involved the balance of the cecal and colonic walls.

RELATION OF BACTERIA TO THE LESIONS

Two bacterial agents, *Salmonella choleraesuis* and *Spherophorus necrophorus*, must be considered with reference to the etiology of the advanced type of necrotic enteritis described.

Spherophorus necrophorus is very widely distributed in nature, being especially abundant in the soil on which animals are held. It is pleomorphic, and individual organisms are from 0.5 to 1.5μ wide and may appear in chains 100 or more μ long. In necrotic tissue of enteritis, Spherophorus may appear as long skeins or coils (Fig. 21.7). The organism is Gram-negative, stains well with polychromatic methylene blue, the latter stain bringing out a beaded appearance. It is nonmotile, does not form spores, and is strictly anaerobic.

Feldman and co-workers (1936) studied

FIG. 21.4—Cecum, ileocecal valve and large colon in S. choleraesuis enteritis. Note swollen wall and diphtheric membrane. (*Biester, Iowa State University*)

er necroses were invariably dependent upon the presence of *B necrophorus* playing, as secondary invader, its role of producer of deep and progressive tissue necrosis

Mohler and Morse confirm these observations by descriptions of sections of intestines from pigs dead of hog cholera

we found in the deeper portions on the border line between the healthy and diseased tissues numbers of bundles of *B necrophorus* In oculation of such material into the back of a rabbit resulted in the usual coagulation necrosis

Biester also isolated *S necrophorus* from the deeper parts of the necrotic enteritis lesions by scraping off the overlying diphtheric membrane under running tap water When the red granulomatous zone was reached, the rinsing was continued with sterile saline solution, and a clean scalpel used to continue the scraping This material was injected subcutaneously or intramuscularly into rabbits In the resulting abscesses *S necrophorus* was usually obtained in pure culture, especially after one or two transfers in rabbits

When poor sanitation faulty nutrition, and other stress factors continue their influence in the presence of *S choleraesuis*, and necrosis of tissue occurs, *S necrophorus* finds conditions favorable for entrance, multiplication, and further invasion into the tissues When *S necrophorus* is firmly established in the deeper parts of the tissue, the invasion and further destruction of tissue continues long after the *S choleraesuis* has ceased its attack In these deeper necrotic zones *S necrophorus* is arranged in long chains and skeins (Fig 21 7) Intestinal lesions from human typhoid fever cases when sectioned and stained for bacteria showed similar masses of organisms in the deeper parts of the process (Fig 21 8) The so called button ulcers of swine present a similar arrangement which must be interpreted as a progressive process caused by *S necrophorus* in the deeper tissues as a secondary invader in a suitable field for growth prepared by a primary agent.

Some field cases of enteritis showed in

flammatory changes in the large intestine with round flattened masses of hard intestinal contents which were adherent to the mucous membrane When these were removed a shallow depression characterized by superficial necrosis appeared On histopathologic examination, dense masses of necrophorus like organisms were found in the underlying crypts (Fig 21 9)

Balantidium coli

This protozoon was found in many cases of enteritis of swine Its presence in the tissues could not be correlated with the occurrence of the disease In some sections of swine intestine, masses of *Bal coli* were found in the caseated tissue and many forms also were seen in the lumen In the material studied *Bal coli* rarely advanced beyond the necrotic or damaged tissue (Fig 21 3) In man *Bal coli* shows marked invasive ability, being found deep in structures that are not necrotic The pig seems to possess considerable resistance to *Bal coli* However, the question of pathogenicity to the pig should be reopened and studied under controlled conditions

PATHOGENESIS OF ENTERITIS PRODUCED BY FEEDING *S CHOLERAESUIS* CULTURE

As the pathological studies of field cases progressed and the relationship of *S choleraesuis* and *S necrophorus* to the lesions were described from different cases, it became evident that the changes seen in the terminal stage of a disease, sometimes extending over a considerable period in some field pigs, might prove misleading unless the various steps in the process could be observed in sequence from inception of the disease For this purpose, a group of healthy pigs were fed broth culture of *S choleraesuis* and then were killed 8 16, 32, 54, 61, 80, 88, 96, 104, 123, 152 187, and 288 hours later (Biester et al, 1925) Necropsies were performed The results of the cecal examinations are given here since the progression of the process as it occurs in the cecum is representative of that in the colon

At 8 and 16 hours these pigs presented of

the occurrence of agglutinins for this or ganism in different species of animals Their data suggest a relationship between the occurrence of agglutinins and the en vironmental conditions of the animals with reference to the intensity and duration of exposure to S necrophorus

Mohler and Morse (1904) also called attention to the ubiquitous character of S necrophorus, its highly infectious nature, and extensive range of pathogenesis They mentioned that Th Smith in examining ulcers for the hog cholera bacilli, found large bacilli following the course of the blood vessels in the granulation tissue un der the sloughed area They noted that Jensen interpreted and associated this de scription with S necrophorus, and that Schutz also found in necrotic areas of the intestine long thread shaped organisms which he named B filiformis Bang, they pointed out inoculated into mice and rab bits necrotic material from the intestines of hogs affected with hog cholera Necrosis developed at the point of inoculation and

in the internal organs Too little considera tion is given to the role of S necrophorus in the production of intestinal necrosis Conditions suitable for invasion of S necrophorus are inaugurated by the initial attack by S choleraesuis The mere bac teriological isolation of S choleraesuis in the absence of detailed pathological studies is largely responsible for the divergent con cepts regarding Salmonella induced en teritis

The earlier observations and interpreta tions regarding the role of S necrophorus were not given their proper place after the discovery of the filtrable virus of hog cholera These facts are clearly stated by Mohler and Morse (1904)

There can be no doubt that hitherto the hog cholera bacillus has been given too much credit for the lesions customarily seen in that disease As long ago as 1887 it was surmised by Schutz and demonstrated in 1889 by Bang and later confirmed by Zschokke Olt and others that the superficial necroses occurring in the intestine and stomach of cases of hog cholera were due to the hog cholera bacillus whereas the deep

FIG 21 7—Cecum Sphe-rophorus necrophorus be neath diphtheric mem brane X 880 (Biester Iowa State University)

no significant changes in the cecum Some of the intestinal contents adhered to the mucosa and a slight degree of edema was observed

32 Hours

The microscopic changes consisted of pronounced desquamation, shallow erosion of the mucosa, and cytoplasmic fragmentation In some sections edema was quite noticeable The outer zone of the mucosa was infiltrated by leukocytes but no exudate appeared in the lumen Gram negative rods simulating *S choleraesuis* appeared in the lumen and at the points of desquamation, together with *S necrophorus*

56 Hours

The cecal wall was swollen At this stage definite patches of caseated membrane appeared Fibrin was abundant at the points of erosion and in the exudate enmeshing the cellular elements

64 Hours

The wall was swollen, edema and cellular infiltration were more advanced than in the tissues of the 56 hour pig, and caseation was progressive and diffuse The exudate on the surface was rough presenting the appearance of the villi of a cows tongue When removed this left depressions in the mucosa Microscopically the mucosal erosion was very marked A membrane composed chiefly of cellular exudate was present on the mucosa which was completely caseated in some places, while in others it appeared in various stages of retrogression Fibrin appeared in the exudate enmeshing the cells and it was arranged in long continuous strands over part of the mucosa Fibrin plays an important role in the formation of these membranes

Sections stained for bacteria showed a mixed flora in the exudate toward the lumen At the epithelial layer or deeper in the mucosal tissue Salmonella like organisms predominated In the deeper parts of the exudate and upper mucosa some

Spherophorus like organisms were present At the point of sloughing the primary and secondary organisms were present together with a mixed intestinal flora

80 Hours

The gross and microscopic changes were more advanced than in the animals killed previously Both mucin and fibrin in the lesions had increased A mixed flora appeared in the upper part of the membrane At the points of mucosal erosions and beneath single organisms and clumps of Salmonella like organisms were found In many crypts short chains of Spherophorus like organisms appeared Apparently anaerobic conditions had not prevailed long enough for the firm establishment of the anaerobic organisms as is noted in field cases of longer duration where Spherophorus organisms appear in dense coils and skeins

88 Hours

This subject showed more advanced changes

96 Hours

A large part of the necrotic membrane had sloughed but microscopic study revealed that the process of necrosis had advanced more deeply than in the subject destroyed 88 hours after the culture was fed

128 Hours

Swelling and edema were progressive and more advanced than in previous cases A marked inflammatory reaction characterized the entire wall The mucosa was covered by a caseated membrane 2 mm thick, which was quite firmly adherent The microscopic picture confirmed the progression of the lesions observed grossly The upper part of the mucosa had undergone caseation necrosis Under this alteration were found extensive karyorrhexis and leukocytosis The balance of the wall showed retrogressive cellular changes a l advanced leukocytic infiltration Extensive an l advanced karyorrhexis and Hemor...
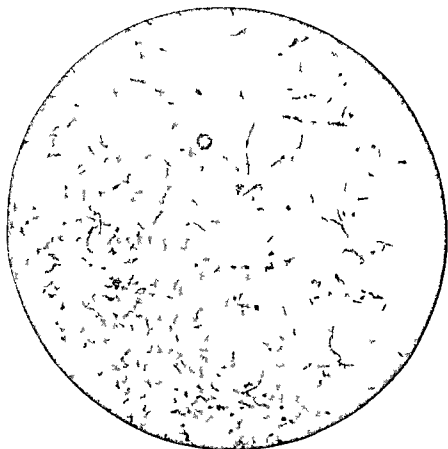
FIG 21 8—Intestinal lesion from typhoid fever of man showing Spherophorus like organisms under case ated membrane X 850 (Biester, Iowa State University)

FIG 21 9—Large colon Spherophorus like organisms in crypt under disc of firm intestinal contents adherent to the mucous membrane X 920 (B ester Iowa State University)

Dorset and his co workers made the same observations under similar conditions when pigs were held at their station

Pigs that were heavily parasitized with ascaris when fed *S choleraesuis* developed more severe intestinal changes than para site free pigs Some pigs died 48 hours after experimental feeding of *S cholerae suis* Upon necropsy these cases always showed invasion of the bile duct or liver by round worms (Biester, unpublished data)

DIFFERENTIAL DIAGNOSIS

Salmonella induced enteritis must be distinguished from enteritis of other causes

Nutritional Deficiencies

The relation of malnutrition to infec tious enteritis has been studied by numer ous workers since 1910 Davis *et al* (1910) observed that small pigs, when fed only corn, developed a necrotic type of en teritis On the basis of this experimentation it was concluded that 100 mg of nicotinic acid daily are required to prevent the de velopment of nutritional enteritis Luecke *et al* (1948) reported that adequate sup plementation of *dl* tryptophane to corn rations prevents nicotinic acid deficiency in the pig and, when 25 per cent casein was added to a corn ration, no symptoms of nicotinic acid deficiency were produced Uncomplicated enteritis due to niacin de ficiency is characterized by an excessive exudate of mucus (Dunne *et al* 1949) Later work by Luecke *et al* (1949) indi cated that deficiencies of niacin panto thenic acid and possibly riboflavin are involved in the production of enteritis Other investigators also produced enteritis by feeding rations deficient in niacin they were unable to isolate *S choleraesuis* from the feces of affected pigs This is not sur prising and must be considered in making correct interpretations Under field condi tions, when lots are contaminated with *S choleraesuis* and the pigs are carriers of the organism, and when nutritional de ficiencies are superimposed both factors must be considered in evaluating the true

situation In all cases of enteritis a careful evaluation of the ration should be made to aid in correct diagnosis and treatment

Enteritis Caused by Vibrio Infections

Enteritis caused by Vibrio infections previously mentioned and sometimes re ferred to as hemorrhagic dysentery, amoe bic like dysentery, and bloody scours was found only twice in Iowa during the studies on enteritis from 1922 to 1927 This form must be considered from a differen tial diagnostic standpoint Sometimes *S choleraesuis* is present in field outbreaks of vibrio caused enteritis In the early stage of the vibrionic enteritis large quantities of red liquid feces containing mucus are passed which have the appearance of cur rant jelly It was because the feces re sembled those passed during amoebic dys entery in man that the vibrionic type of enteritis was referred to as amoebic like dysentery by Kinsley, although amoeba were not present in the cases studied by Biester and co workers (1935) Transpor tation by truck which has facilitated the movement of swine direct from farm to farm or through public sales barns, has in creased the occurrence of the more severe and acute types of infectious enteritis The vibrionic form of enteritis now is more common in Iowa than it was 30 years ago *S choleraesuis* was not isolated from the two cases of acute hemorrhagic dysentery found during the studies on enteritis from 1922 to 1927 in Iowa From the remaining cases not characterized by the passage of red blood *S choleraesuis* was recovered During the summer of 1934 a field out break of acute hemorrhagic enteritis of the vibrionic type was studied *S choleraesuis* was not recovered from any of the pigs or specimens from this herd (Biester *et al*, 1935) Passages by feeding intestinal con tents and small pieces of intestinal wall and transmission by pen exposure were continued for about 4 months The test pigs used were animals raised on the Vet erinary Research Institute area They had been held on clean pasture were healthy subjects and did not carry Salmonella

found in the submucosa The wall
:ared to have lost its functional ability

Hours

his subject showed a very characteristic
nment of the aerobe and anaerobe In
ologic sections stained for bacteria Sal
iella like organisms and a mixed flora
e seen in the upper part of the mem
ne whereas in the deeper parts only
erophorus like organisms were noted
'ecrosis extended deeper into the mu
152 hours after *S choleraesuis* culture
fed In the 176 hour pig the muscu
s mucosa was obliterated by necrosis
aghing of the necrotic membrane in
ces changed the alignment of the two
anisms of primary interest

The changes produced in the large colon
e comparable to those described in the
um (Fig 21 10) *S choleraesuis* was
lated from the lymph nodes of the pig
itroyed 32 hours after the culture was
l

The factors which reduce the resistance
the pig may predispose invasion of the
body by *S choleraesuis* Repeated obser
vations on field herds in Iowa over a
period of 12 years showed that, where poor
sanitary conditions prevailed, the nutri
tional status was equally poor When such
pigs were moved from the lots that had
held pigs for long periods and were placed
on clean pasture, the intake of bacteria and
parasitic agents was diminished and the
nutritional status was improved

RECOVERY WITHOUT MEDICATION

During the course of the investigation,
groups of pigs with enteritis which were
scouring and passing pieces of necrotic
membrane, were purchased and brought to
the Veterinary Research Institute, where
they were placed in clean pens having
concrete floors The pens in which these
pigs were held pending subsequent ne
cropsy were washed several times each day
When some of these pigs were held in
reserve for considerable periods, they im
proved and some ultimately recovered and
grew to market weight The ration in
cluded a considerable amount of bran
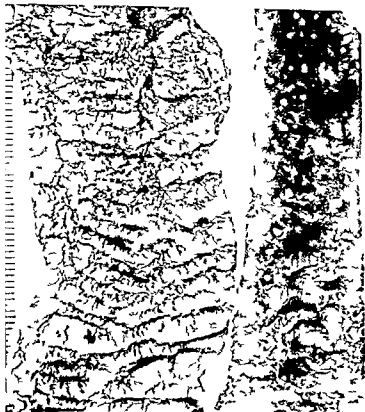
FIG 21 10—Experimentally pro
duced *S choleraesuis* enteritis Large
colon—advanced deep necrosis and
diptheric membrane Small colon—
advanced hemorrhage and focal
necrosis (Biester Iowa State Uni
versity)

cases of septicemia in the abattoir were pas saged through rabbits and through media to eliminate the possible presence of hog cholera virus They call attention to the role of predisposing factors and to the en hancement of virulence by continued pas sages through pigs The latter condition would be fulfilled if pigs were held on the same lots for long periods, especially with poor drainage or sanitation

Rasmussen et al (1911) experimentally produced enteritis in swine by feeding cul tures of S choleraesuis When the culture was mixed in the feed, the reaction was more severe than when it was given as a drench No difference was observed in the mortality rate among pigs fed different rations

Van Es (1946) has very ably documented and evaluated the divergent results and interpretations on the pathogenicity of this organism

Schoening et al (1919) reviewed some of the concepts regarding the status of S choleraesuis as a primary pathogen They present evidence that both cultures pre served by lyophilization retain their patho genicity for the pig Gwatkin and Moyni han (1944, 1915) met with varying suc cess in their attempts to produce necrotic enteritis by feeding a number of strains of S suipestifer under artificially induced stress Guthrie (1952) produced a form of enteritis by feeding a virulent culture of S choleraesuis which had been lyo philized

CONTROL AND TREATMENT

The early history of treatments for swine enteritis follows the general pattern of treatments for human typhosis Some of these, including copper sulfate alkaline agents, soaked medicated whole oats but termilk and salt, have been replaced

Edgington et al (1942) found that nico tinic acid was not a preventive for enteritis which was induced by feeding cultures of S choleraesuis

The results of studies on nicotinic acid as a specific or preventive for enteritis are

not conclusive Krider (1942) summarized this well

Recent work indicates that necrotic enteritis in swine is not solely a nutritional disorder Ne cro may still be regarded as a bacterial disease and diet may serve to prevent or permit the de velopment of the organism Sanitary measures are recommended to hold the causal organism in check This possibly may be as important in the control of necro as the role of nicotinic acid

Rasmussen et al (1914) concluded that the use of nicotinic acid during the re covery period of survivors produced bene ficial results However, they recommend the administration of a composite of B vitamins instead of nicotinic acid alone Cameron (1942) experimentally treated field cases of enteritis with sulfaguanidine administered in gelatin capsules at the rate of 1 gm per 20 lb body weight 4 times daily for 5 days He reported some relapses 6 to 8 days after treatment was stopped The economic factor would pre clude this procedure for field use

Kernkamp (1945) reports favorable re sults by the use of 0 75 to 1 5 gm of sulfa guanidine per 10 lb body weight in one dose on the first day and on the succeeding 5 days, half the dose to be given in the morning and half in the evening He also used sulfasuxidine 1 0 to 1 5 gm per 10 lb body weight 6 or 8 days Improved sanitation to avoid reinfection is also recommended

Edmonds (1948) administered parenter ally in field cases of enteritis 3 gm of sulfa merazine per 85 to 100 lb body weight daily Sulfathalidine at the rate of 1 gm per 10 lb body weight was also used He considered these drugs to be of value in enteritis pneumonia associated with the presence of S choleraesuis

Sulfathalidine has been used *per os* in daily doses from 0 25 to 0 50 gm or more per 10 lb of weight It is not readily ab sorbed in the intestinal tract which is a favorable characteristic in the treatment of enteritis

Boley et al (1951) claimed good results in treating an outbreak of dysentery as

Many of these experimentally infected pigs died but S choleraesuis was not recovered from the internal organs throughout the investigation Streptococci or coli form organisms were isolated occasionally from pigs that succumbed during the night and which were not examined until the next morning However, in some cases of vibrio enteritis, S choleraesuis may be present. If the disease (vibrionic or hemorrhagic type) does not kill the animals, blood disappears from the feces and from the lesions in the intestines After the free blood and tenacious mucus disappear, a diphtheric membrane forms In the later stage this lesion is indistinguishable from that found in advanced cases of S suipestifer enteritis The severe destruction of tissue, as a result of the initial vibrio damage in subjects that do not die, often is followed by invasion and multiplication of S necrophorus, the end result is similar to the gross changes which follow primary injury from S choleraesuis invasion The appearance of gross blood in the feces during the early stage distinguishes this type of enteritis from that caused by the invasion of S choleraesuis under conditions of body stress In the experimentally produced S choleraesuis cases the temperature rise begins 24 hours after the culture is fed whereas in the hemorrhagic form (vibrio) the temperature rise appears from the fourth to the sixth days and diarrhea is manifest on the sixth day following the feeding of the infectious material The elimination of blood usually follows 1 or 2 days after the onset of diarrhea in the vibrio induced cases Salmonella enteritis diarrhea may appear from 1 to 4 days following administration of the culture

Coccidiosis

Coccidia may be present in S choleraesuis enteritis or in enteritis caused by vibrio and their presence should not be overestimated in differential diagnosis The several species of coccidia from swine which have been studied in pure culture under controlled conditions do not produce necrotic or hemorrhagic changes The epithelial cells parasitized with coccidia may be destroyed in situ, may desquamate, or may migrate to a subepithelial position leaving a denuded mucosa None of the species found in swine involve the deeper tissues Considerable mucus may be present in the feces but any hemorrhage that is present may be attributed to causes other than Eimeria Isospora suis is of low grade pathogenicity

S CHOLERAESUIS AS A PRIMARY PATHOGEN

The ubiquity of S choleraesuis and the unsuccessful efforts of some investigators to produce a necrotic form of enteritis have produced divergent views regarding the status of this organism After taking cognizance of the possible effect of the pathogenicity of the cultures fed, nutritional status of the test pigs, sanitation, parasitoses, and other stress factors involved the record shows substantial evidence supporting the view that S choleraesuis under proper conditions assumes the role of a primary pathogen In support of this view are the results of Salmon and Smith (1886a, 1886b, 1887, 1889), Dorset et al (1905), Murray et al (1927) Glasser (1927) notes that necrotic forms of enteritis were described by Roloff in 1863 and 1875 and these probably were due to Salmonella infections These cases occurred in young pigs that were closely confined under unsanitary conditions and poor husbandry, and when these untoward conditions were corrected, the disease disappeared from the premises Glasser concludes from his observations that this form of enteritis is based upon a predisposition caused by poor management, confinement in a damp environment, faulty nutrition (especially calcium deficiency), together with the constant intake of feces containing the organism Biester et al (1928) repeatedly produced enteritis by feeding cultures of S choleraesuis Hindmarsh and Edgar (1933) experimentally produced septicemia and extensive lesions of necrotic enteritis in swine by feeding broth cultures of S choleraesuis These cultures originating from

EDMONDS, E V 1918 Sulfamerazine and sulfathaladine for enteritis pneumonia syndrome in swine Vet Med 43 460, and chart

FELDMAN, W H, HESTER, H R AND WHERRY F P 1936 The occurrence of Bacillus necrophorus agglutinins in different species of animals Jour Infect Dis 59 159

FOURNIER, J, DE LAJUDIE, P, AND BARGOO E R 1953 Enquete sur les salmonelles chez les porcs a Saigon Ann Inst Pasteur 81 792

GALTON, M M, LOWERY, W D, AND HARDY A V 1954 Salmonella in fresh and smoked pork sausage Jour Infect Dis 95 232

——— SMITH, W V McELRATH, H B AND HARDY A V 1954 Salmonella in swine cattle and the environment of abattoirs Jour Infect Dis 95 236

GLASSER, K 1927 Die krankheiten des Schweines 3rd ed M & H Schaper Hannover, p 222

GUTHRIE, J E 1952 Nitrofurazone as a therapeutic agent in S choleraesuis enteritis of young swine Vet Med 47 307

GWATKIN, R, AND MOYNIHAN, I W 1941 The effect of diet chilling and gastro intestinal irritation on susceptibility with S suipestifer Canad Jour Comp Med 8 66

——— AND ——— 1915 Attempts to infect swine with S suipestifer cultures and necrotic material from the intestinal tract Canad Jour Comp Med 9 71

HINDMARSH, W L, AND EDGAR, G 1933 Some observations on B suipestifer septicemia of pigs Vet Res Rep No 6 Part III (Dec 1931—Dept of Agr New South Wales) p 126

HORMAECHE, E, AND SALSAMENDI, R 1939 El cerdo normal como portador de salmonelas Arch Urug Med 14 375

HUNTOON, F M 1918 Hormone medium—a simple medium employable as a substitute for serum medium Jour Infect Dis 23 169

KERNKAMP, H C H 1945 Gastroenteric disease in swine Jour Amer Vet Med Assn 106 1

KRIDER, J L 1942 Swine nutrition No Amer Vet 23 769

LUECKE, R W, McMILLEN, W N, THORP, F JR, AND TULL, CAROLYN 1918 Further studies on the relationship of nicotinic acid tryptophane and protein in the nutrition of the pig Jour Nutrit 36 417

——— THORP, F JR, McMILLEN, W N DUNNE H W AND STAFSETH, H J 1919 A study of B vitamin deficiencies in pigs raised on farms Mich Agr Exp Sta Tech Bull 211

LUTJE, F 1938–39 Beobachtungen uber die Haufigkeit des Vorkommens von Enteritis bakterien beim Schwein zugleich ein Beitrag zum Kapitel der Fleischvergiftung Deutsch tierarztl Wschr 46 798, 820 47 13

McBRYDE, C N 1937 Pneumonia in swine resulting from Salmonella suipestifer infection No Amer Vet 18 (June) 41

MOHLER, J R, AND MORSE, G B 1904 Bacillus necrophorus and its economic importance U S D A Bur Anim Ind 21st Ann Rep p 76

MURRAY, C, BIESTER, H E, PURWIN, P, AND McNUTT S H 1927 Studies on infectious enteritis of swine Jour Amer Vet Med Assn 52 34

RASMUSSEN, R A, STAFSETH, H J, FREEMAN, V A, AND MILLER, M J 1944 Influence of B vitamins liver and yeast on induced necrotic enteritis in swine Vet Med 39 421

ROSSOFF, I S AND ABEL, W 1953 Bacitracin Its use in control and treatment of swine enteritis Vet Med 48 369

RUBIN, H L, SCHERAGO M AND WEAVER, R H 1942 The occurrence of Salmonella in the lymph glands of normal hogs Amer Jour Hyg 36 (1) July 43

SALMON, D E, AND SMITH, T 1886a The bacterium of swine plague Amer Monthly Microbiol Jour 7 204

——— AND ——— 1886b Investigations in swine plague U S D A Bur Anim Ind 2nd Ann Rep p 184

——— AND ——— 1887 Investigations of swine diseases U S D A Bur Anim Ind 3rd Ann Rep, p 20

——— AND ——— 1889 Nature and prevention of hog cholera U S D A Bur Anim Ind 4th and 5th Ann Rep, p 63

SAPHRA, I, AND WASSERMAN M 1954 Salmonella choleraesuis A clinical and epidemiological evaluation of 329 infections identified between 1940 and 1954 in the New York Salmonella center Amer Jour Med Sci 228 525

SCHOENING, H W, DALE, C N, MOTT, L O, AND HABERMAN, R T 1949 Pathogenicity of S choleraesuis Maintaining virulence by lyophilization Amer Jour Vet Res 10 101

DE SCHWEINITZ, E A, AND DORSET M 1903 A form of hog cholera not caused by the hog cholera bacillus U S D A Circ No 41 Bur Anim Ind, p 1

TEN BROECK, C 1918 A study of paratyphoid bacilli isolated from cases of hog cholera Jour Exper Med 28 759

UHLENHUTH, P, MESSNER H AND GEIGER, W 1929 IX Viruschweinepest Vol 9 Handbuch der pathogenen Mikroorganismen Kolle W Kraus R and Uhlenhuth P Gustav Fischer und Urban Schwarzenberg Jena

VAN ES, L 1946 Pig Typhus (Salmonellosis suis) Univ Nebr Agr Exp Sta Res Bull 147

sociated with both *Vibrio coli* and Salmonella types by the administration of 100,000 units of bacitracin *per os*

Rossoff and Abel (1953) incorporated bacitracin in ground feed at the level of 50 gm per ton to treat enteritis Guthrie (1952) controlled the disease by 1 gm of nitrofurazone on the first day and 0 5 gm on the second day He recommends that in some cases treatment should be continued at lower levels in the feed

Bruner (1956) expresses the view that drug or antibiotic treatment of salmonellosis may ameliorate the condition but they do not cure or eliminate carriers He recommends surroundings and management which will avoid body stress, and sanitation

Streptomycin, chlortetracycline, oxytetracycline, and other antibiotics have been used in treating various types of enteritis The administration of these materials as well as vitamins and sulfonamides should be considered only as supportive measures and not as specific treatment Relapses have been reported in connection with the use of many of these medicaments when the administration was discontinued Unless the underlying factors which made possible invasion of the tissues by Salmonella and Spherophorus are removed or corrected, therapeutic treatments will fail The re covery of enteritis cases when the animals are held in clean pens without medication supports this view Microscopic examination of the intestines of these recovering subjects reveals rapid regeneration of the mucosal epithelium The present trends and practices in holding pigs on concrete or dry lots place dependence on antibiotics and other feed additives to "take care of the disease level" These are poor substitutes for a balanced ration of unadulterated feedstuffs and clean pastures properly managed Troughs and waterers should be moved frequently to avoid the development of wallows around their locations When lot sanitation is neglected, ultimately the antibiotics and feed additives fail to prevent losses Lot rotation dilutes the parasitic ova and infectious agents eliminated by the animals, thus minimizing reinfection Salmonellosis, like many other infectious diseases, spreads more rapidly when animals are closely confined and held in large groups The nutritional status of the animals is improved after they are placed on clean pasture This also aids in recovery from the infection

REFERENCES

Biester, H E., Murray C. McNutt, S H, and Purwin, P 1928 Studies on infectious enteritis in swine II The pathogenesis of infectious enteritis Jour Amer Vet. Med Assn 72(7) 1003
———, Schwartz L H, and McNutt, S H 1955 General consideration of enteritis in swine—more than one type based on etiology Iowa Vet 6(5) 5, 22
Bolley, L E Woods, G T, Hatch, R. D And Graham, R 1951 Studies on porcine enteritis Cornell Vet. 41 251
Breed R S., Murray, E. G D, and Smith, L. L. 1948 Bergey s Manual of Determinative Bacteriology 6th ed Baltimore Md p 508
Bruner, D W 1956 Salmonella presented for identification during the 5 year period of 1950-51 Cornell Vet. 46 11
———, and Edwards P R., 1940 Ky Agr Exp Sta Bull 404
———, and Moran, A. B., 1949 Salmonella infections of domestic animals. Cornell Vet 39(1) 53
Cameron, H S., 1942. Field investigations on sulfaguanidine in swine enteritis Cornell Vet 32 1
Dale, C N , Meriwether, H., and Schoening, H W 1944 A *Salmonella choleraesus* bacterin phage in swine feces Amer Jour Vet. Res. 5 279
Davis, G K., Freeman V A. and Hartman, L. L 1940 The relations of nutrition to the development of necrotic enteritis in swine. Mich. Agr Exp Sta Tech Bull No 170 p 23
Dorset M., Bolton B M, and McBryde, C. N 1905 The etiology of hog cholera U.S D.A Bur Anim Ind Bull 72 p 1
Dunne, H W , Luecke, R. W., McMillen W N., Gray, M L., and Thorp, F, Jr. 1949 The pathology of niacin deficiency in swine. Amer Jour Vet. Res. 10 351
Edgington B H., Robison, W L., Burroughs, W., and Bethke, R M., 1942 Tests with nicotinic acid for the prevention of infectious swine enteritis. Jour Amer Vet Med Assn 101 103

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Swine Erysipelas

Swine erysipelas, or its equivalent in other languages — Schweinerotlauf, rouget du porc, antrace eresipelatoso, and erisipela del cerdo — is a specific disease of swine caused by the bacterium Erysipelothrix rhusiopathiae. Although the word erysipelas (red skin) is loosely descriptive, it does have a universal meaning For this reason, such synonyms as diamond skin disease, urticaria, nettle rash, and other symptomatic terms could well be eliminated from general usage

Swine erysipelas is worldwide in distribution and is a serious economic disease of swine throughout Europe, Asia, and the North American continent In South Africa, South America, New Zealand, and Australia it is of limited economic significance

HISTORY

The identification of swine erysipelas as a disease entity began in 1878, when Koch isolated an organism from an experimental mouse which he called "the bacillus of mouse septicemia" The similarity of this organism with that causing Schweinerotlauf was pointed out by Loeffler in 1881 Pasteur and Thuillier in 1882 83 briefly described an organism isolated from pigs with rouget and prepared a vaccine for use in pigs against this disease In 1885, Loeffler presented the first accurate description of the causative agent of Rotlauf, or rouget, and described the infection in swine Rosenbach in 1887 isolated the organism from affected humans (erysipeloid) but considered it to be different from the

cause of mouse septicemia and swine erysipelas Kelser and Schoening (1943) mentioned that the three species, rhusiopathiae, muriseptica, and erysipeloidis, are now generally considered to be identical

In the United States, the history of swine erysipelas began with the isolation by Smith (1885) of an organism from the kidney of a pig which resembled that causing rouget, or swine erysipelas Moore (1892) recovered a similar appearing organism from the spleen of a pig during the year 1888 Smith (1895) isolated the swine erysipelas, or mouse septicemia bacilli, from swine tissue originating in Minnesota At that time he said, ' it is not improbable that this bacillus may gain enough virulence to produce epizootics, if such is not already the case, and that in endeavoring to trace the causes of swine diseases a search for the swine erysipelas bacillus should not be neglected "

No subsequent reference to this disease was made until Tenbroeck (1920) reported finding Bacillus murisepticus in the tonsils of swine Creech (1921), by his recovery of the erysipelas organism from cutaneous "diamond skin' lesions, again pointed to the presence of this swine disease in the United States Ward (1922) found B erysipelatis suis in a large percentage of arthritic joints obtained from an abattoir. Further, he induced arthritis in a pig by intravenous inoculation and recovered the organism in pure culture from an affected joint Ward, referring to arthritis, stated that ' the lesions studied are typical of those observed with great

frequency in the principal hog slaughtering centers of the United States"

Giltner (1922) isolated the organism from a three week old pig dying of acute swine erysipelas Parker et al (1924) demonstrated the direct association of the organism with diamond skin lesions, necrotic dermatitis, polyarthritis and evidence of septicemia in market hogs During 1927 according to Smith (1955), Foster man in South Dakota, called attention to a new serious disease of hogs The cause of this disease was shown to be *B erysipelatis suis* in 1930 by Breed (1933), and this was confirmed by the work of Taylor (1931) Harrington (1933) reported the presence, from 1929 to 1932, of acute erysipelas in seven states By 1937, the erysipelas organism had been identified as a cause of disease in 28 of the 48 states (Breed 1937) The increasing seriousness of erysipelas can be followed in Reports of the Committee on Transmissible Diseases of Swine, U S Livestock Sanitary Association The American Veterinary Medical Association Committee on Diseases of Food Producing Animals, in 1952, reported this disease of swine to rank first in four states, second in eight, and third in three states During the 10 year period 1942 51, swine erysipelas was responsible for an estimated annual loss of $21,000,000 [1]

NATURAL HOSTS

Jenn (1931) pointed out that in Europe the erysipelas organism had previously been recovered not only from swine but from pigeons, mice, guinea pigs, lambs cows, colts and from the bone marrow of a dead horse Kondo and Sugimura (1931) isolated the organism from both marine and fresh water fish, houseflies, and rotten horsemeat. Van Es and McGrath (1936), in a review of European and American literature, reported that the organism had been found also in man, dog duck, fowl, turkey, mud hen, parrot, common sparrow, canary birds, finches siskins, thrushes, blackbirds turtledoves, and quails Levine

(1952) mentioned the recovery of the organism from pheasant, peacock, wild mallard, parakeet, white stork, and a herring gull Of interest in the United States, Wayson (1927) found in Southern California an epizootic caused by *B murisepticus* among meadow and house mice Ray (1931) isolated organisms similar to *Ery rhusiopathiae* from the joint of a lamb and indicated that other recoveries had been made as early as 1924 Marsh (1931) also isolated *Ery rhusiopathiae* from the affected joints of lambs Beaudette and Hudson (1936) identified the organism as responsible for a disease outbreak in a turkey flock Erysipelas in turkeys has since become a bacterial disease of major importance to the turkey industry [2] Graham et al (1939) reported heavy losses in ducklings, which were apparently due to *Ery rhusiopathiae* The organism has been associated with death losses in chickens (Breed, 1943, Grey, 1944, Evans and Narotsky, 1954) Stiles (1944) recovered the erysipelas organism from a wild brown rat Hartsough (1945) isolated the organism from farm raised mink Moulton et al (1953) observed arthritis in calves, and the joints of one calf yielded the organism of erysipelas Connell (1951) noted its presence in a northern chipmunk Seibold and Neal (1956) isolated *Ery rhusiopathiae* from a dead porpoise, which had been living in captivity

Klauder s (1938) review of erysipeloid in man serves to emphasize the varied sources of infection These ranged from the handling of swine carcasses and their by products in the abattoir to the handling of fish, clams, tallow, grease, fertilizer, pelts and a horse carcass Morrill (1939) isolated the erysipelas organism from a lesion on the hand of a veterinary student, as well as from a poorly preserved portion of a horse carcass which the student had been dissecting

SUSCEPTIBILITY OF
EXPERIMENTAL ANIMALS

White mice and pigeons are highly sus

growth in plain broth after 24 and 48 hours' incubation at 37°C is best described by Smith (1885), who noted "a faint opal escence, which, on shaking, was resolved for a moment into delicate rolling clouds Slight sedimentation will be seen after 24 hours' incubation After 48 hours, upon gentle shaking of the tube in a circular motion, the viscous appearing sediment will slowly spiral upward, forming a tail Breed et al. (1948) gave the optimum pH for growth as 7 6 The addition of serum to fluid media results in a more luxuriant growth In studying the growth require ments of two strains of the organism Hut ner (1942) found the necessary materials to be one or more amino acids supplied as a casein or gelatin hydrolysate, ribofla vin, and oleic acid as a substitute for serum

Byrne et al (1952) observed that some strains produced a small zone of alpha hemolysis around smooth type colonies, although they found this factor was not constant with a strain and was more pro nounced and regular with rabbit than with horse blood They observed also that rough colonies did not induce hemolysis

Biochemical Reactions

According to Karlson and Merchant (1941), *Ery rhusiopathiae* produces hydro gen sulfide, but tests for indol formation nitrate reduction, catalase formation, and methylene blue reduction were negative The Voges Proskauer and methyl red tests also were negative Byrne et al (1952) re ported that nitrate reduction and produc tion of hydrogen sulfide can be variable It has been demonstrated by Usdin and Birkeland (1949) that strains of the organ ism are capable of producing hyaluroni dase Rowsell (1955) studied the hyalur onidase activity of recent isolates and ones kept on artificial medium for a year, and found no qualitative difference in their ac tivity

In carbohydrate media, according to Breed et al (1948), acid but no gas is formed in glucose, galactose, fructose, and lactose and is formed more slowly in man nose and cellobiose Neither acid nor gas is produced in arabinose xylose, rhamnose, maltose melibiose, sucrose trehalose, raf finose, melezitose dextrin, starch, inulin, amygdalin salicin, glycerol erythritol, adonitol manitol, sorbitol, dulcitol, or in ositol The addition of serum can result in an acid reaction in dextrin, maltose, mannose, sucrose and trehalose Inocula tion of litmus milk may result in little or no indication of acidity It is recognized that the fermentation reaction of this or ganism can be somewhat variable, which fact is well discussed by Byrne et al (1952) Wix and Woodbine (1955), and Tiffany (1955) For this reason, familiarity with the general pattern of fermentation reactions of known strains in the media routinely used would be advantageous

Serological Characteristics

Marsh (1933), Barber (1939), and Julianelle (1941) found strains of *Ery rhusiopathiae* to be serologically similar Watts (1940) divided them into two serological groups He found that each group possessed a heat stable specific anti gen, and in addition, each probably con tained two common heat labile antigens which are present in different proportions, and are responsible for cross agglutination Atkinson (1941) divided Australian strains into two distinct antigen groups and a third intermediate group Gledhill (1945) showed that strains may be classified ac cording to their predominant antigen and proposed to refer to these as Groups I, II and III, with Group IV comprising strains which did not fall within these groups Gledhill (1947) demonstrated that growth in serum media produced both thermo in serum media produced both thermo stable O antigen and a labile L antigen He suggested the possibility of killed OL suspensions being used in the active im munization of pigs against swine erysipelas Dedie (1949) described two serological variants A and B of *Ery rhusiopathiae*, which were characterized by a variant specific acid soluble antigen Some strains, called N forms did not possess these anti

SMOOTH

X 900

X 48

INTERMEDIATE

X 900

X 42

ROUGH

X 900

X 43

FIG. 33 *Leptospira* photographies. Photography by R. E. Scherr. Courtesy A.B.S.
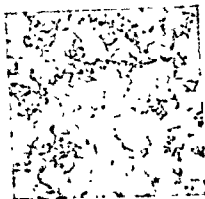
manner in which they move conveys the impression that they are in pain When walking, they manifest either a rather stiff stilty gait or obvious lameness Upon stopping, they may be seen to shift their weight in an apparent effort to ease the pain in their legs If left alone, they will soon lie down, but do so carefully The pigs show ing disinterest may evidence some reluctance at being disturbed, but will make little or no effort to get away Upon being forced to get up, they may stand for only a few moments before lying down again While standing, the feet are carried well under them and the head is hung dejectedly, giving the back line a marked arched appearance Others will not be able to stand and even when assisted will wobble or stagger and eventually sit down or fall over The manifestation of arthritis may remain and progress to permanent damage of the affected articulations As the ani mal recovers, it can also either disappear permanently or recur and become chronic

Most animals will show either a light appetite or complete inappetence Oc casionally an animal will back away from the feed and regurgitate Bowel move ments are usually retarded and the feces firm and dry, although as the disease progresses a diarrhea may appear Harrington (1933) observed that diarrhea was common among small pigs but rarely seen in old hogs

Cutaneous lesions (urticaria, or dia mond skin lesions) appear as early as the second and usually by the third day after exposure to infection They are described by Munce (1942) as resembling insect bites On the light skinned hog they can be seen as small, light pink to dark purple areas that usually become raised, are firm to the touch, and in most instances are very easily felt by running the hand over the shoulders, back, sides, and belly In ani mals with dark pigmented skin one must rely mainly on palpation, although when observed from a proper perspective, the weltlike lesions can be seen on the lesser haired areas of the skin At times, the hair over a lesion along the back and sides will

be slightly raised above the surrounding level The lesions may be few in number, and thus easily overlooked, or so numerous it would be difficult to count them all Individual lesions by extension of the bor ders assume a shape which is quite char acteristic when seen or felt for they easily suggest a square, rectangular or rhom boidal pattern These lesions may, in a few days gradually lose their swelling and coloration and disappear, with no subse quent effect other than a superficial de squamation to mark the site In other in stances the lesions become joined losing their individual identity and cover large areas of the skin The intensity of these skin lesions which seems to be indicated by the degree of coloration from light pink to an angry purplish red has a direct re lationship to the outcome of the infection Light pink to light purplish red lesions will disappear within four to seven days after their first appearance whereas the extensive angry dark purplish red lesions can precede either death or necrosis of the skin thus passing into a chronic manifes tation of the disease The difference be tween the early appearance of diffuse coloration and the coloration resulting from the joining of individual skin lesions as compared with the presence of a few scattered lesions, appears to be one of de gree only, in that the conditions which control infectivity (susceptibility of the host, virulence of the organism, and the route of infection) permit a sudden over whelming multiplication of the organism with dispersion throughout the body, as contrasted with the same process but which occurs in a milder or, for want of a better term, a less explosive manner (Fig 22 3)

In addition to the clinical significance of the cutaneous or urticarial lesions, Grey (1917a) demonstrated the important re lationship of these lesions with the presence of the causative organism in body organs Of 61 packing house hogs showing urti carial lesions, 29 showed alterations of the internal organs, indicating a septicemic condition, whereas 32 showed no visible alteration and were considered normal

gens He found living cultures of both A and B groups capable of inducing good immunity After killing the organism, only those of B group produced immunizing vaccines This immunity protected mice against groups A and B Rice *et al* (1952a) in a study of Canadian strains noted, as did Gledhill (1945), that such differences as were observed between strains appeared to be quantitative rather than qualitative

Ewald (1955) examined what he called the O (serological complete) and o (dissociated variant) forms of three strains of organism of antigenic groups A, B, and N, and found that dissociation was accompanied by a loss of group specific antigen and a reduction in agglutinogenic activity

Dinter (1948) reported on the specific hemagglutinating properties of a strain of *Ery thusiopathiae* Dinter and Bakos (1948) demonstrated that only a few ery sipelas strains show good hemagglutinating properties They believed the factor in a strain for hemagglutination and that for producing immunity are identical Schell ner and Schleibheim (1949), following tests on horses used for producing hyper immune serum, found evidence that a strain of high hemagglutinative ability produces immune serum with a high inhi bition titer They mentioned it was not clear whether the degree of inhibition parallels the protective ability of the serum Roots and Venske (1952) observed that all B strains had hemagglutinating proper ties and that A strains did not have this ability The work of Radvila (1953) indi cated that all hemagglutinating strains autolyze spontaneously but others do not, and autolyzed cultures adsorbed on alumi num hydroxide immunized mice better than those which are not prone to autoly sis Gelenczei and Rappay (1955) noted that living B strains produced, within a shorter time in horses, a more potent serum than did the A strains

Increased awareness of the antigenic dif ferences of erysipelas strains has helped in the development of biologics, and appears to have been an aid in the pro duction problems associated with speci fic immune serum of equine origin It may well be that a better understanding of these strain differences will help serve to explain more fully the nature of this disease as it is encountered in the field

CLINICAL SIGNS

The clinical signs of swine erysipelas can be divided into three general headings acute, subacute, and chronic Although visible signs demand the most attention one must recognize also the existence of subclinical or inapparent infection

Acute Erysipelas

Acute swine erysipelas is characterized by its sudden appearance, with death of one or more animals Other animals in the herd may be noticeably sick, and a few of these may subsequently die Those visibly sick will have temperatures of 104° F and over, and those with the more extreme temperature may show signs of chilling The height and variable persistence of temperature will follow fairly well the course of the disease Harrington (1933), however, reported temperatures of around 106° F, and Railsback (1951), tempera tures up to 109° F, although the animals still appeared normal (Fig 22 2)

Affected animals withdraw from the herd and will be found lying down While some may remain alert, others show signs of complete disinterest The former, when ap proached, will resent being disturbed but will get up and move away This usually will be accompanied by squealing, and the

FIG 22 2—Acute swine erysipelas, field case Note discoloration of right ear and parts of the body (Courtesy Dr L Van Es)

FIG 22 5—Chronic swine erysipelas
Left, normal joint Right, affected
joint (Courtesy A R S , U S D A)

erysipelas is the result of some degree of
the acute infection

PATHOLOGICAL CHANGES

With the exception of individual cu
taneous lesions, acute erysipelas presents
no macroscopic changes of a strictly pathog
nomonic nature at necropsy The lesions
that are observed are those of septicemia
In the light skinned animals, there is usu
ally seen a purplish red discoloration of
all or portions of the snout, ears, throat,
abdomen, and legs Incision of the skin
lesions reveals vascular congestion and
purplish red discolorations of the sub
cutis The lungs may be congested and
edematous Ecchymotic hemorrhages are
generally seen on the epicardium over the
auricles In the abdominal cavity, the
stomach and small intestines frequently
show a slight or marked inflammation that
may be either catarrhal or hemorrhagic
The liver is usually markedly congested,
and the gall bladder either normal or
somewhat shrunken in appearance Of par
ticular note is the appearance of the spleen,
for it may be congested and markedly en
larged, and, according to Hutyra et al
(1938) and Van Es and McGrath (1942),
punctiform hemorrhages may be present in
the cortex of the kidneys The gross ap
pearance of the lymph nodes will depend
upon the degree of involvement in the
area they drain When affected, they may
appear either edematous and enlarged or
show marked peripheral congestion The
mucosa of the urinary bladder either may
be normal in appearance or may present

areas of congestion Affected joints may
show an increase in the amount of synovial
fluid This fluid may be viscous or sero
sanguinous and inflammation of the intra
articular tissue may be observed

A histopathological examination of the
skin showing diffuse purplish red discolor
ation reveals damage to the capillaries The
pathologic changes occur in the papillae
and the upper layers of the derma The
blood vessels of the papillae are congested
and distended with blood and may contain
organisms suggestive of Ery rhusiopathiae
The papillae may also present lymphoid
cell infiltration and focal necrotic areas as
a result of circulatory stasis Collins and
Goldie (1940) described an arteritis in the
animals they examined that resembled
polyarteritis nodosa and rheumatic arter
itis in man There is initial swelling, hya
line degeneration of the media, and peri
vascular infiltration by lymphocytes and
fibroblasts These changes are seen in the
heart, kidney, mesentery, and synovial
membanes Satoh et al (1953) made sim
ilar observations Godgluck and Wellmann
(1953) noted these degenerative changes
in the blood vessels of the papillae of the
corium of pigs showing acute septicemia
following experimental exposure Affected
lymph nodes usually show acute hyper
plastic lymphadenitis, and the cell poor
substance of the nodes showing congestion
contain erythrocytes, accounting for the
gross hyperemia Some investigtors de
scribed changes in the kidneys as hemor
rhagic nephritis or as glomerulonephritis,
while Satoh and co-workers preferred to

bacteriological examination of the spleen, liver, and representative lymph nodes revealed that 86 per cent of the hogs classified as septicemic and 78 per cent of those classified as normal yielded *Ery. rhusiopathiae.*

Subacute Erysipelas

Subacute erysipelas includes symptoms which are less severe in their manifestations than the acute. The animals do not appear as sick, temperatures may not be as high or may not persist as long; appetite may be unaffected; a few skin lesions may appear which may be easily overlooked; and, if visibly sick, the animals will not remain so for the same length of time as those acutely ill.

Chronic Erysipelas

Chronic erysipelas follows the acute infection and is characterized by necrotic changes, which involve loss of portions of the skin, ears, tail, and feet, valvular changes in the heart, and, of most importance, the occurrence of arthritis.

The areas of necrotic skin are dark, dry, and firm, and eventually become separated from the healing underlying tissue and fall off, leaving an ugly scar (Fig. 22.4). Secondary infection usually occurs and slows the healing process, which, at best, extends over many weeks.

Localization of the infection on the heart valves can give rise to symptoms of cardiac insufficiency and will be most noticeable following exertion.

Chronic arthritis results in joints that show various degrees of stiffness and enlargement (Fig. 22.5). Interference with locomotion ranges from slight to complete, depending upon the extent of damage and number of joints involved. Van Es and McGrath (1942) indicated that arthritis may occur as an independent manifestation of erysipelas. Aitken (1950) has observed apparently healthy pigs in affected herds that later develop arthritis in spite of treatment. Railsback (1951) and McNutt (1954) were of the opinion that all chronic

FIG. 22.3—Cutaneous or urticarial lesions of swine erysipelas. (Courtesy A.R.S., U.S.D.A.)

FIG. 22.4—Chronic swine erysipelas, field case. (Courtesy Dr. Floyd Cross.)

areas of skin pigmentation, should serve to differentiate these conditions The loss of the tail, portions of the ears, and even the phalanges, can be a sequela of erysipelas infection However, there are other causes of these conditions such as injury, frost bite, and bacterial infections other than *Ery rhusiopathiae* Peterman (1944a), with the assistance of A G Beagle, has prepared a chart in the form of lists of various clinical symptoms requiring yes or no answers which serves as a useful guide for the differential diagnosis of this disease

ARTHRITIS

Chronic erysipelas as represented by arthritis has been recognized for many years, however, the association of *Ery rhusiopathiae* and arthritis in swine on a national scope is sometimes questioned Arthritic swine constitute an important economic problem in the industry because the condition not only affects the rate of growth of hogs, but is responsible for the loss of edible meat and increased cost of packing house operations A summary of activities by the U S D A Meat Inspection Branch for the fiscal year 1956 shows arthritis ranking third as a cause for swine carcass condemnation Additional losses include not only parts of carcasses but also the downgrading of hams and shoulders which became mutilated during the course of final inspection The subject of arthritis in swine is also of academic interest in the field of human medicine because of its similarity to rheumatoid arthritis in man (Collins and Goldie, 1940, Doyle, 1949 Hughes, 1953)

The work of Ward (1922) and of Parker et al (1924) called attention to the presence of the erysipelas organism in polyarthritis of market hogs Stiles and Davis (1934) isolated the organism from approximately 30 per cent of arthritic joints originating in the abattoir A review of the literature by Collins and Goldie (1940) and the results of their investigations illustrated further the relationship of *Ery rhusiopathiae* and arthritis of swine Gwatkin (1940) examined bacteriologically a

series of joints from a packing house and found the erysipelas organism in 50 per cent of them Grey et al (1941) and Osteen (1941-42) examined about 1,000 arthritic joints of market hogs and recovered the erysipelas organism in approximately 75 per cent of them Connell et al (1952) identified the organism in 36 5 per cent of the arthritic joints examined The joints of eleven 10 week old pigs affected with periarthritis yielded *Ery rhusiopathiae* (Jansen et al, 1956)

Localization of infection in the joints and surrounding tissue by streptococci, staphylococci, cornyebacteria, and Brucella can be responsible for arthritis and accompanying lameness Swan (1949) associated pellagra with a secondary infection in young pigs which led to a crippling arthritis Collier (1951) identified five beta hemolytic streptococci obtained from cases of suppurative arthritis McNutt et al (1948) and Switzer (1951) isolated a filterable agent from the affected joints of pigs which may prove to be of more significance than is now realized

Other causes of joint abnormalities are injury and the absence of a proper nutritional balance or adequate assimilation of the mineral elements in the feed Border line rickets is a common cause of lameness in growing pigs If the rachitic process is permitted to continue, the animals may develop enlarged joints and arched backs Holm et al (1912) observed, during a dark, cloudy spring period lameness in two litters that quickly responded to vitamin D therapy Earle and Stevenson (1952) found that high phytic acid content of cottonseed meal was primarily responsible for a lameness in growing pigs, and more calcium, phosphorous and vitamin D in the feed were required to prevent the condition In addition, dietary deficiencies of copper, riboflavin, pantothenic acid, pyridoxine, and choline cause lameness and stiffness according to Beeson et al (1953)

Despite the recognized causes of joint abnormalities other than *Ery rhusiopathiae*, and in the absence of a comparable survey of these causative factors, the

refer to the changes as hemorrhages. They also observed one instance of parenchymatous degeneration of the adrenal gland. Sikes *et al.* (1955a) in their experimental series noted that the adrenal gland was enlarged in some chronic cases, and the zona glomerulosa was *infiltrated by many* leukocytes. The blood picture in acute erysipelas is one of mononuclear leukocytosis with a neutrophilic leukopenia.

Animals affected with the chronic form of the disease present an enlargement of one or more articulations. There may be a gangrenous process involving the skin, ears, tail, and phalanges; and vegetative proliferation on the heart valves is not uncommon. The internal organs may show evidence of chronic inflammatory changes, and if there are growths on the heart valves they may reflect the lesions that accompany passive hyperemia.

Granulation tissue forms within the affected articular cavity with proliferation of connective tissues, thus forming elongated tags that may be suspended in the joint fluid or attached to the synovial membrane. There is thickening of the capsular ligaments and induration of the periarticular structures. There may be erosion of the articular cartilages along with periostitis and ostitis. Ankylosis of the involved joint by fibrous adhesion may also be accompanied by calcification.

Vegetative growths on the heart valves are composed of granulation tissue and superimposed masses of fibrin. Connective tissue proliferation occurs with additional fibrin formation which, in time, interferes with normal heart function and can also be the source of emboli. As Kernkamp (1911) pointed out, however, all instances of this lesion affecting the heart valve are not necessarily caused by *Ery. rhusiopathiae*, and streptococci are capable of producing a similar lesion.

DIAGNOSIS

Clinical

Swine erysipelas in its acute form cannot readily be differentiated from other septi-

cemic diseases such as hog cholera, acute salmonellosis, and primary bacterial infections in young animals. A history of sudden deaths within a herd, several sick with high temperatures, variable appetite, stiffness and lameness, spontaneous recovery, and/or subsequent development of chronic lameness with visible joint malformation, are symptoms presumptive of swine erysipelas. The recognition of characteristic square or rhomboid-like skin lesions is the only conclusive diagnostic finding. Internally, the presence of an enlarged spleen is suggestive of erysipelas. With regard to punctiform hemorrhages or petechiae of the kidneys, McNutt (1954) considered them to be of much more significance in hog cholera since they appear in 90 to 95 per cent of such cases.

Swine erysipelas in the chronic form, as represented by necrosis and desquamation of areas of the skin, may be confused with severe sunburn, photosensitization (Fig. 22.6), the effect of ectoparasites, and parakeratosis. Careful attention to the herd history, nature and location of the skin lesions, and their relation to light and dark

FIG. 22.6—Photosensitization or rape poisoning, field case. Note necrotic portions of the ears and skin over the neck. (Courtesy Dr. L. T. Railsback.)

Connell and Langford (1953) who placed the tissue to be cultured into broth which was then held under refrigeration for several days The resulting fluid inoculum was then plated onto a somewhat modified Packer s medium, incubated for 24 hours at 37° C, and held for another 24 hours at room temperature before examination of the plates Erysipelas organisms may also be recovered from white mice or pigeons following death, which have been injected with either tissue fluid or the contaminated culture Although the organ ism may be present in the material used for injection, the mice or pigeons may not die because of either too few numbers or the relative avirulency of the organisms In this regard, Wellmann (1954b) found that organisms recovered directly from ar thritic joints and lesions on the heart valves had little pathogenicity for mice and pigs but after growth on artificial media they generally acquired high virulence for these animals

Preliminary identification of the organ ism is based on (1) selection of several suggestive appearing single colonies with the aid of either a hand lens (10X) or broad field microscope and inoculation into nutrient broth (2) characteristic growth in broth after 24 and 48 hour incubation (3) morphological appearance w h e n stained according to Gram, and (4) the reaction pattern in carbohydrate media For final confirmation a mouse protection test is conducted If the unknown is *Ery rhusiopathiae*, the mice receiving only the culture should die, while those receiving the culture and specific immune serum should remain alive and healthy If the unknown is avirulent for mice, the serum of rabbits can be tested, after a series of inoculations, for the presence of specific agglutinins using *Ery rhusiopathiae* plate antigen A strong agglutination reaction will confirm the identification

Serological

The tube and plate agglutination test was introduced by Schoening *et al* (1932) Their investigations (1935 and 1936) and the findings of Stiles and Davis (1934) and

Rice *et al* (1952b) have shown the test to have definite limitations, and the best diagnostic results were obtained in chronic cases of swine erysipelas Since the test is more applicable to a herd than to the in dividual diagnosis, several animals should be tested to get a representative picture Grey *et al* (1941) have described in detail a method for preparing the antigen and a technique for conducting the test

Wellmann (1955a) reported on the ex perimental application of the hemagglu tination inhibition test (HI) and the wachstumsprobe (WP) or growth test, as a means of testing the pig for susceptibility and/or immunity He recommended that the two tests be conducted together in or der to determine the status of the animal In subsequent experiments, Wellmann (1956) has noted the tests work differently in different age groups and in those pigs that became immune through the colos trum of immune dams, by vaccination, or by natural or artificial infection

IMMUNITY

According to Friedberger and Frohner (1908), the first effort to immunize ani mals against swine erysipelas was made by Pasteur (and Thuillier) in 1882, who used organisms of reduced virulence In 1892 Lorenz introduced the active passive method, involving first the use of immune serum and followed in a few days with an injection of culture Leclainché, in 1897 proposed that the serum and culture be mixed and administered simultaneously A later modification of these methods re sulted in the simultaneous administration of serum and culture, and this procedure has been followed for over 50 years Traw inski (1919) has reviewed briefly the many efforts in the past to improve the prophy lactic control of swine erysipelas Hruska (1952) developed a colloidal hydrolyzed antigen (bacterin), and mentioned also the immunizing properties of bouillon cul tures treated with brilliant green, ethyl glycol, and crystal violet in mice and pigeons Delpy and Hars (1953) reported on the preparation of a lysed vaccine (Im munogene)

findings of Grey et al (1941) strongly in
dicate that the erysipelas organism must be
considered responsible for the majority of
arthritic hogs in this country

Bacteriological

Recovery of *Ery rhusiopathiae* from the
living or dead animal provides a definite
diagnosis of erysipelas Hubbard (1952)
and Brudnjak and Kralj (1955) reported
on the use of hemocultures as a diagnostic
aid in the living animal A single pig
however, may have a negative hemoculture
one day and may be positive on one or
more succeeding days For this reason,
hemocultures should be made on several
affected animals in the herd

Tissue for bacteriological examination
should be handled in a manner so as to
reach the laboratory in the best possible
state of preservation Sterile cotton tip ap
plicators in individual glass tubes can be
conveniently used to obtain inoculum dur
ing the necropsy When possible, the heart,
lungs, liver, spleen, kidney, lymph node (s),
affected joint (s), and a section of skin and
underlying tissue (4" x 4") showing dis
coloration should all be examined This is
because the organisms may be few in num
ber or seemingly absent in some of the tis
sues and may be very numerous and easily
recovered in others Thus, the omission of
certain tissues may account for the absence
of laboratory confirmation of the field
diagnosis Morrill (1945) noted from
laboratory examinations that the kidney
yielded the organism most frequently with
spleen, liver, and affected joints following
in that order The importance of examin
ing several tissues was well demonstrated
by the findings of Muller (1954), who
examined bacteriologically 15,891 pigs re
quiring emergency slaughter *Ery rhusio
pathiae* was found in 4,139 of them Of
this number, the organism was present in
the musculature and organs of 1,829, in
several organs of 1,563, kidney only of 336,
spleen only 280, liver only 84, lymph nodes
only 20, musculature only 16, and in mis
cellaneous parts in 11 pigs In addition, in
one series of 1,152 positive findings, 404 or
35 per cent of the cases were submitted

without any history of erysipelas being
suspected

Joints are more easily opened, and in a
sterile manner, when one end is secured
in a vise The skin, underlying muscle,
fatty portions, and loose pieces of tissue
are first removed Then, with a Bunsen
burner, the surface is thoroughly seared
The joint is opened with a flame sterilized
scalpel using leverage obtained either by
grasping the proximal extremity with the
fingers protected by several thicknesses of
paper towel or by inserting a heavy pair of
forceps, or the like, into the marrow cavity
Several inoculations, consisting of synovial
fluid, bits of intra articular tissue and
scrapings of affected cartilaginous surface
should be made into culture media Af
fected joints with varying degrees of visible
alterations within the joint cavities do not
necessarily assure the isolation of *Ery
rhusiopathiae* or other bacteria Sikes et al
(1955a) were unable to isolate the organ
ism in cases showing advanced chronic
arthritis 32 weeks after exposure Con
versely, Hughes (1955) found the organism
in joints up to 83 weeks after experi
mentally induced arthritis

The affected skin section must be ap
proached from below, for this reason it
should be removed leaving a smooth un
dersurface which can be more easily seared
by the Bunsen burner After searing, a
convenient sized 'well' is then made by
removing a section of tissue down to the
epidermis using flame sterilized scalpel and
forceps, carefully avoiding puncturing the
relatively thin epidermis By making shal
low crisscross incisions and scrapings with
the scalpel, tissue fluid and debris are made
available for the inoculation of media

Isolation of *Ery rhusiopathiae* from tis
sue or cultures containing organisms of
spreading colonial nature is sometimes de
sirable A simple and practical method was
found by Dale (1940) which consisted of
transferring a loopful of culture into 0 25
per cent phenolized broth and incubating
at room temperature for 24 to 18 hours
A loopful of this culture is then streaked
on solid medium for subsequent examina
tion Another method was developed by

The gilts were found to be immune from 4 to at least 8 months after vaccination When weanling pigs were vaccinated and tested at intervals for immunity by the percutaneous method of challenge, 58 per cent of the pigs were found to be protected up to and through the average marketable age of 5 to 7 months (Shuman, 1953b). In an effort to increase the number of protected pigs, Shuman and Earl (1954) vaccinated weanlings with an arbitrarily selected dosage ratio of 1:6, (culture ½ ml. and serum 3 ml.), in place of the prescribed ratio of 1:20, (culture ¼ ml. and serum 5 ml.). The results of percutaneous exposure indicated a marked improvement in the number of pigs protected and the duration of immunity after vaccination. Thus, on an experimental basis, simultaneous vaccination of weanlings appeared to be a useful procedure in providing protection for pigs to the time they are marketed.

Although the experimental results from gilt and weanling pig vaccination readily suggested a cyclic vaccination schedule, there is no reported experimental or field evidence to show that such a procedure is of value. It is quite possible that, instead of the culture stimulating a booster effect, resistant weanling pigs tend to neutralize its immunizing potential (Shuman and Earl, 1954).

Attenuated or Avirulent Vaccines

Vaccines of reduced virulence have been in use for many years in Europe and Asia; the principle behind their application originated with Pasteur. In 1955, a vaccine of this general description was licensed and made available to veterinarians in the United States, although its use within a state was subject to the discretion of the respective State Livestock Sanitary authorities.

Reduction in virulence or attenuation of selected strains of the organism entering into the production of the principal vaccines have been accomplished through exposure to either air-drying (Staub, 1939,

1910), or to media containing acridine derivatives. Kondo et al. (1932) used trypaflavine and rivanol, and subsequently introduced what is known as the Kondo vaccine (Kondo and Sugimura, 1935). Sandstedt and Lehnert (1944) also used trypaflavine to develop the Swedish AV-R9 vaccine.

Kondo and Sugimura stated that trypaflavine-fast organisms were not virulent for mice and pigs, and the latter have tolerated up to 800 ml. without harmful effect. Wiidik (1952), and Staub mentioned the attenuated organism is "practically" avirulent for mice and is nonpathogenic for pigs. Of the Swedish vaccine, Wiidik (1955) observed that it did not cause a local or systemic reaction in pigs, even with a 50-ml. dosage, and that the organism had not been recovered from them following vaccination. Further, no difficulty had been encountered following its use in bred gilts and pregnant sows during the first half of gestation. Gray and Norden (1955) demonstrated the avirulency of a domestic product in mice, pigeons, guinea pigs, turkeys, swine, and man. Of Staub's vaccine, Trawinski (1949) concluded from field tests in Poland that it was permanently attenuated and had lost its pathogenic properties for pigs; the incidence of post-vaccination trouble was due to "the presence of erysipelas carriers among pigs."

Trypaflavine-fast vaccines do not recover their original virulence after repeated transfers on favorable media or through animal passage. Parnas et al. (1954) stated that, following the passage of Staub's avirulent strain in bouillon containing swine serum, there appeared a rise in virulence.

The means by which these avirulent vaccines induce immunity has been explained by Wiidik (1952), and Murase and Shimizu (1953) from their work with mice. Wiidik observed there was a limited multiplication of the organism in the animal's body soon after injection, and they were found in the spleen and lymph nodes 100 days later. In other words, multiplication

The biological control of swine erysipelas can be grouped under four headings (1) serum, (2) serum plus virulent culture or the simultaneous method, (3) attenuated or avirulent vaccines, and (4) killed or ganisms or bacterins

Serum

Serum is obtained from horses which have been hyperimmunized through re peated inoculations of the erysipelas or ganism Normal pigs injected with serum receive immediate passive immunity, but it is of relatively short duration It is gen erally believed the immunity persists for about two weeks

Serum and Culture

The vaccination of swine in the United States, using virulent culture and hyper immune serum of equine origin, began in Nebraska in 1936, under the guidance of Van Es and McGrath In 1938, a coopera tive state and federal project for the con trol of erysipelas was initiated State authorities designated the areas in which the culture vaccine could be used and the veterinarians who were permitted to apply it This arrangement served to limit the indiscriminate use of the vaccine in all parts of the country In 1956, 27 states were participating in the cooperative program

The results of a 10 year study involving more than 12 million animals were sum marized by Peterman et al (1948) With regard to safety, they observed the number of cases of erysipelas following vaccination were extremely small, however, they stressed the importance of using only live culture where the disease is known to exist, and where it poses an economic threat Vaccinated animals did not appear to seed down the premise nor to transmit the dis ease to non vaccinated animals Neither were they harmfully affected when preg nant, or when vaccinated simultaneously, with hog cholera serum and virus On the basis of a one year study, they concluded that an additional injection of culture alone had no advantage over the one

simultaneous vaccination with serum and culture When vaccinated and non vac cinated herds in erysipelas areas were com pared, the evidence indicated that im munization in pigs was produced following vaccination, and that it was of sufficient degree and duration to protect the great majority during the time the pigs were on the farm The duration of immunity on an average persisted for 6 months They recommended that a control program could be started at any time regardless of age and size, but preferred to vaccinate the gilts after they had been selected for breed ing purposes, and their sucklings before they were 2 weeks of age The dosage of serum was as follows 5 ml for pigs up to 50 lbs, 10 ml from 50 to 100 lbs, 15 ml from 100 to 150 lbs, and 20 ml for over 150 lbs Culture dosage was ¼ ml for each 5 ml of serum In this connection, Van Es et al (1940), Van Es and McGrath (1942) and Fechner (1954–55) have pointed out that satisfactory immunization with this method depends upon a balance between the quantity and potency of the serum and the infective potential of the culture Standards have been developed for the pro duction of these biologics, although these standards have been related to pigeons or mice In actual practice the amount of cul ture and serum used is quite variable, and there is a need for re examination of the dosage schedule in pigs

Shuman (1953a), using the percutaneous method of challenge, observed that the vaccination of baby pigs from an erysipelas infected premise with serum and culture 2 to 5 days after farrowing was of no value in protecting growing pigs Because ery sipelas is not uncommon in sows and suck ling pigs, and even in pigs a few days of age as was noted by Waller (1938), some prophylactic measure is necessary during these intervals of life Shuman (1953c), following the suggestion of Peterman et al (1948), found baby pigs derived from gilts which were immunized through vaccina tion, to be immune to percutaneous ex posure up to and including 6 weeks of age

after 21 to 28 days In field trials conducted from 1947 to 1951, Maas (1953) noted the single vaccination produced protection in pigs at most up to 3 months when vaccinated twice, there was an adequate immunity for at least 6 months These results were comparable to those experienced by investigators in other parts of Europe according to Maas

Under laboratory conditions and using the percutaneous exposure technique Thomson and Gledhill (1953) found the single injection of bacterin provided protection for at least 10 weeks Windik and Ehlers (1953) observed pigs to be immune at 3 weeks but at 10 weeks 8 of 19 vaccinated pigs showed evidence of generalized infection The work of Shuman (1954) indicated a variability between two commercial products, with the better of the two inducing 100 per cent immunity at 1 month, 28 per cent at 2 months, and 16 per cent at 3 and 4 months after a single vaccination Gouge et al (1956b) found the single injection to protect over one-half of the pigs 3 months after vaccination and demonstrated the superiority of the double over the single vaccination At 4 months after vaccination those double treated showed over 70 per cent protection, as against approximately 25 per cent of the single treated pigs Sikes et al (1956) using an intravenous challenge route, observed that some protection was evident 6 weeks after vaccination, but mentioned it was not complete because all the pigs developed chronic polyarthritis

Delpy and Hars (1953) introduced a lysed vaccine (Immunigène) In general, their technique consisted of allowing maximum multiplication of the organisms, the addition of a bacteriostatic, and followed by an undisclosed method which encouraged autolysis An adjuvant, saponin, was added to the product to produce local irritation and delay absorption after vaccination A product produced in this manner was licensed and made available to veterinarians in the United States in 1955

This product must be injected subcutaneously at the junction of the ear and head, and in the exact dosage indicated by the producer (Delpy, 1955) He also mentioned that, due to the saponin, when injected into the skin, it will cause necrosis if in the muscle, a strong inflammatory reaction will result Ordinarily a local soft tender swelling will appear soon after vaccination This swelling will decrease in size after 2 days leaving a firm almond sized nodule

Using the intradermal exposure technique pigs withstood 1,000 D E (equal to 10 million X minimum reacting dose) 8 months after vaccination In the field, Delpy (1955) stated, 'after 12 months some vaccinated pigs may show a variable degree of skin reactivity, but these pigs cannot as far as we can see, be infected by contact with sick animals'

TREATMENT

An erysipelas serum (Susserin) was first recommended as a curative in 1899 by Schutz and Voges, although Lorenz in 1892 introduced the use of immune serum from pigs horses, and sheep in connection with prophylactic vaccination [6] For approximately the next 50 years, hyperimmune serum was the only worthwhile available form of treatment Its value and limitations have been noted by Harrington (1933), Lentner (1940), Munce (1940), Van Es and McGrath (1912), and Aitken (1950) Although affected animals often recover quite quickly after serum therapy, others do not respond favorably and may die or develop chronic manifestations of the disease For maximum effectiveness the serum must be administered early in the course of the disease The dosage, injected either subcutaneously or intravenously, generally depends upon the weight of the animal and will vary from 10 to 10 ml or more

Porter and Hale (1939) observed that sulfanilamide had no therapeutic effect in treatment of mice inoculated with *Erysiopathiae* Klauder and Rule (1941) and Konst (1915) reported discouraging

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[*] Cited by Friedberger and Fröhner, 1903.

impractical level of stress on the degree of immunity of the vaccinated animals, and the individual differences in the interpretation of the results may convey an erroneous impression of the relative merits of a biologic for swine erysipelas.

EPIZOOTIOLOGY AND CONTROL

Experimental Methods for Inducing Erysipelas in Swine

The most outstanding problem confronting investigators in the past was the inability to reproduce swine erysipelas experimentally with consistency. Nevertheless, Fortner and Dinter's review (1944) of parenteral experimental transmission experiments from 1885 to 1942 in Europe and the United States, as well as their own contribution, served to emphasize that erysipelas in varied form had been reproduced by many individuals. The works of Ward (1922), Murray,[7] Watts (1940), Collins and Goldie (1940), and Schoening et al. (1940) also add to the accumulated evidence. More recently, the successful application of exposure methods for the study of experimental infection in swine have been reported by Shuman (1951), Usdin et al. (1952), Hars and Delpy (1953), Godglück and Wellmann (1953), Wellmann (1953), Spencer (1954), Cooper et al. (1954), Nishimura et al. (1954), Sikes et al. (1955a), Rowsell (1955), Hughes (1955), and Gouge et al. (1956a).

Swine erysipelas can be induced experimentally in susceptible swine by the introduction of virulent organisms into the body of the animal, either orally or by several parenteral routes; however, of the various routes, the percutaneous or skin scarification and intradermal methods have an advantage in that, in addition to animal attitude and temperature, they permit visible estimation of the severity of the infection as measured by the character of the cutaneous reaction (Figs. 22.7 and 22.8).

The percutaneous or skin scarification
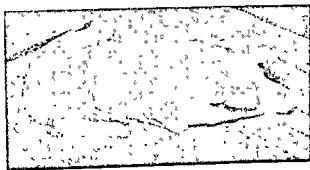
FIG. 22.7—Acute swine erysipelas, experimental infection. Note discoloration of the ears, jowl and right hind leg. (Courtesy A.R.S., U.S.D.A.)

method of Fortner and Dinter (1944) consists of placing on the previously clipped side of a pig, at equal intervals, vertical lines of scarification. A desirable degree of scarification is reached when a slight amount of serosanguineous fluid appears uniformly along the line. By means of cotton-tipped applicators, the organisms in a fluid medium are inoculated into the abraded skin. In the author's experience, approximately .07 ml. is applied to each line of scarification 5 to 6 inches in length. Further, in one experiment, there was no particular difference in the development of cutaneous reaction between lines on the same pig that received an estimated 7,000, 70,000, 700,000, or 7,000,000 organisms. In another experiment each inoculated line

FIG. 22.8—Chronic swine erysipelas, experimental infection. Note necrotic portion of the skin and beginning of sloughing. (Courtesy A.R.S., U.S.D.A.)

results on the therapeutic value of sulfon amide compounds A review by Wood bine (1950) of his work and others per mitted the conclusion that the sulfonamide compounds which they examined are in effective in the treatment of erysipelas in fection

Heilman and Herrell (1944), Van Es et al (1945), and Grey (1947b) demon strated the bacteriostatic action of penicil lin salts *in vitro* and *in vivo* Schatz and Waksman (1944) and Woodbine (1947) reported streptomycin to be effective *in vitro*, the latter finding it to be less ef fective than penicillin Prier and Alberts (1950) observed penicillin to be more ac tive *in vitro* than chlortetracycline and streptomycin Moynihan and Stovell (1954), following *in vitro* and *in vivo* tests, found *Ery rhusiopathiae* sensitive to chlortetracycline, oxytetracycline, and tetra cycline and highly sensitive to penicillin G Streptomycin was of doubtful value even at dosages four to five times that of penicil lin Erythromycin, bacitracin, and chlor amphenicol were ineffective *in vivo*, al though erythromycin showed marked *in vitro* activity against the organism Wix and Woodbine (1955) by their tests, showed that streptomycin, dihydrostrepto mycin, chloramphenicol, polymyxin B, and neomycin were without antibacterial ac tivity, oxytetracycline, chlortetracycline, and bacitracin, in this order, had decreas ing antibacterial activity, penicillin and magnamycin had continued antibacterial activity over a period of 7 days, the former being considered the most satisfactory anti biotic at this time

The successful application of penicillin in the treatment of swine erysipelas was first reported by Aitken (1919) His sug gested dosage was about 1,000 units of penicillin in about ⅓ ml of serum for each pound of body weight, and recom mended the animals be observed in 12 to 24 hours Wiebner (1952) found pigs treated with penicillin showed a recovery rate of about 95 per cent, with an average recovery time of 24 hours, whereas with only serum the recovery rate was about 79 per cent, with an average recovery time of 3 days

Muller (1955) treated pigs with peni cillin at the rate of 3,000 to 4,000 units per kilogram of body weight, which was ad ministered twice in 24 hours, and experi enced 89 per cent recoveries, whereas serum alone gave 42 per cent recoveries In addition, 28 per cent of the pigs treated with serum and 5 per cent treated with penicillin showed residual effects of ery sipelas later When serum and penicillin were combined, he reported that 88 per cent of the pigs recovered, 8 per cent died, and 4 per cent required emergency slaugh ter

Railsback (1956) recommended the use of procaine penicillin G in oil plus serum the former being administered at the rate of 3,000 units per pound of body weight, and the latter varying from 5 ml for baby pigs, 20 ml for pigs 75 to 100 pounds to 40 ml for those over this weight If after 18 hours an animal has a temperature of over 104° F, the treatment may be repeated All treated pigs are ear notched for per manent identification

Evaluation of Biologics

Field evaluations serve a necessary and useful purpose, however, the results are subject to controversy because of such fac tors as (1) passive and active immunity through natural causes, (2) the difference in induced levels of immunity in individ ual pigs through vaccination, (3) the vari ability of exposure to infective organisms from none at all to one of high patho genicity, (4) the wide variation of clinical symptoms presented by erysipelas, and (5) in most instances the absence of non vac cinated controls On the other hand, lab oratory examinations can eliminate most of the variables and permit the accumulation of accurate data. At the same time, the data are limited in their application in that the conditions of an experiment can only approximate the conditions under which the biologic operates on the farm The route and degree of challenge selected may be unnatural and impose a high but

erysipelas was not conducted Of 280 pigs challenged for susceptibility by either a multiple or a single intracutaneous or sub cutaneous injection, 100 failed to show evi dence of disease

Friedberger and Frohner (1908) stated pigs were least predisposed to erysipelas during the first months of life, and that it rarely occurs after 3 years of age They also mentioned that the susceptibility of differ ent breeds of pigs varies greatly, and noted the "common country pig" was the least susceptible Age related to insusceptibility may be explained from two standpoints (1) passive immunity in the young and (2) active immunity in the older animals Suckling pigs of immune dams were shown to be immune to infection several weeks after birth (Shuman, 1953c) In addition Wellmann (1955a) believed this period of passive immunity may extend to 12–16 weeks of age when the dam is affected with chronic arthritis Older animals can acquire immunity following prior subclinical or in apparent infection Illustrative of this was the percutaneous exposure of 15 normal appearing sows originating from a known erysipelas infected premise, of which 13 were found to be immune (Shuman, 1953a) Harrington (1933) could see no relationship on the farm between the dis ease and particular breeds of pigs

Fortner (1947, 1953) discussed inherent resistance to disease and demonstrated, in swine, a family resistance to infection Ani mals do present varying degrees of suscep tibility to experimental infection, although in regard to Fortner s selection and breed ing of resistant pigs, Wellmann (1955a) at the same institute, demonstrated that the resistance' was due mainly to the presence of passive immunity and/or the result of subclinical disease in the pigs

Michalka (1939) stated the body be comes hypersensitized against *Ery rhusio pathiae* by its presence in the tonsils or in testinal tract and that re exposure to the organism provokes an allergic organic re action in the form of arthritis, endo carditis, or skin necrosis This theory has not been confirmed or disproved and it

may enter into the mechanics of some form of the disease process Nevertheless, an al lergic state is not essential in order to in duce experimental erysipelas in pigs

Doyle (1947) demonstrated that pigs previously immunized with crystal violet hog cholera vaccine and later challenged with regular virus, have a greatly increased susceptibility to experimental infection This may embody a principle not yet un derstood, but again it is not an essential condition in order to reproduce the disease Cooper *et al* (1954) and Sikes *et al* (1955a), following the principle of Doyle, could see no difference between prepared and unprepared pigs in their reaction to experimental infection In this connection, Morrill (1945) found no definite relation ship between the incidence of swine ery sipelas in pigs over 2 months of age which had been inoculated against hog cholera, and that in pigs which had not been inocu lated

Kobe (1943) stated it was impossible to infect healthy pigs but when the organism was associated with the virus of infectious gastroenteritis, visible evidence of the dis ease was produced Erysipelas can be as sociated with other swine diseases never theless, it can also be a primary infection and in this sense does not require the pres ence of some other infectious agent for its disease producing ability

Factors that affect the physiological state of the animal s body, such as nutrition sanitation, atmospheric conditions, and sea son of the year, have long been linked to the appearance of the disease on the farm Hutyra *et al* (1938) considered the prin cipal predisposing causes were fatigue, sudden changes of diet, excessive fattening, and exposure to cold They also stated the enzootic appearance of the disease in the summer months was dependent on condi tions of high temperature Trautwein (1949) listed feed containing little salt and vitamins, and little calcium and no vita mins Aitken (1950) noted a relationship between the occurrence of the disease and the eating of an animal carcass (as did

of scarification was immediately rubbed perpendicular to the line with a gauze pad, and it was noted that the cutaneous reactions which followed paralleled those of the control pig

The intradermal injection method of exposure, using a series of graded doses of the inoculum, was first applied in pigs by Watts (1940) Hars and Delpy (1953) reported on its application for the titration of vaccines and immune sera against swine erysipelas Their technique consists of making tenfold dilutions of the exposure strain and injecting into the skin 0 2 ml of each dilution with the object of determining the minimum reacting dose Intradermic injections can be made using a 27 gauge, ⅜ inch needle (Shuman, 1951) On susceptible pigs, characteristic individual cutaneous lesions will develop at the inoculation site Hars and Delpy have observed that, theoretically, 10 organisms were sufficient to produce a local reaction

Factors of Susceptibility

Failure to reproduce the disease experimentally in pigs has generally been related to (1) varying virulence of the organism, (2) age of the pigs, (3) natural resistance of the animals to infection through a genetic influence, (4) allergic or organic state, and (5) disease complex These factors have been advanced also with relation to the natural occurrence of the disease on the farm In addition, such factors as (1) nutrition, (2) sanitation, (3) atmospheric conditions, and (4) seasonal effect have been associated with susceptibility and the natural occurrence of erysipelas on the farm

Schoening et al (1940) induced a fatal infection in pigs by the inoculation of splenic material and a culture from the spleen of a pig dying of the naturally occurring disease In a later unreported experiment, additional pigs were injected with material from the same source and no visible symptoms of erysipelas were observed The questions were raised whether the failure was due to a loss of virulence of the organisms, or whether the pigs were resistant for some unknown reason It has been known for many years that strains of the organism vary in virulence, which fact was again demonstrated by Gouge et al (1956a) in pigs with strains from different sources, both foreign and domestic Obviously, in order to produce disease, the organisms must have some degree of virulency Another requirement is susceptible pigs In this connection, Peterman (1944a) postulated that past failures to reproduce the disease may have been due to the selection of pigs which had already acquired an immunity without showing effects of the disease It has been shown that pigs develop immunity following percutaneous infection (Shuman, 1951) Wellmann (1955a) demonstrated subclinical infection in pigs induced by exposure to strains of low pathogenicity, also resulted in immunity to subsequent percutaneous exposure with virulent organisms In the field it is recognized that the manifestations of erysipelas vary from the acute to the chronic, and that it also can exist on a farm in low grade form and be unrecognized For this reason, erysipelas in subclinical form, or at least in an inapparent form, could account for the presence of one or more naturally immune pigs Thus failure to induce the disease experimentally in the past could have been due principally to the selection of pigs already immune This is illustrated by the following experiences Harvey[8] selected normal pigs from a farm where there had been no history of erysipelas, yet a group of these animals did not react to percutaneous infection with organisms of known ability to induce cutaneous and systemic reaction In the work of Sikes et al (1955a), it was noted that of 100 pigs which were parenterally exposed 28 remained healthy Their pigs were obtained from farms in Indiana that did not appear to have been troubled by erysipelas Jarvis (1956) selected pigs from several farms in northwestern Nebraska where erysipelas was not a problem, and where vaccination for hog cholera and

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[8] Cited by Shuman 1953a

showed the organism to be present in the feces and urine of affected pigs,[11] this information also indicated a means by which the disease was disseminated More recently, Rowsell (1955) demonstrated oral feeding of susceptible pigs induced the infection, which in turn produced shedding of the organism in the feces Whether the organism can invade normal mucosa has not been shown, however, Olt[12] postulated it gained entrance through lesions produced by intestinal parasites

Natural infection undoubtedly can result from infected wounds of the skin This possibility was suggested by Jensen and demonstrated experimentally by Fritsche[13] When one considers how easily infection through the skin can be accomplished experimentally and how prone pigs are to superficial abrasions of the skin under natural conditions, it seems most likely that infection in this manner would not be uncommon

The possibility of insect vectors transmitting erysipelas under natural conditions cannot be overlooked Experimentally, Wellmann (1949) demonstrated that the stable fly, *Stomoxys calcitrans*, could transmit the disease to mice and pigs after feeding on artificially infected pigs He also presented evidence that the common house fly, *Musca domestica*, was capable of transmitting infective material (1955b) As was mentioned previously, Kondo and Sugimura (1934) were able to isolate the organism from the common housefly Stryszak and Oyrzanowska (1955) demonstrated that the mouse sucking louse, *Polyplax serrata* strain, could transmit the infection from sick to healthy mice

Stability of the Organism

Ery rhusiopathiae is considered quite resistant to environmental influences Under artificial conditions, according to Van Es and McGrath (1942), the organisms are killed in 4 days at 111° F, 15 minutes at 125° F, 10 minutes at 131° F, and in a few minutes at between 131 and 137° F They also noted that some variations in the resistance to heat have been observed Dale (1940) found a strain of the organism to be viable in hog cholera virus blood for a period of at least 99 days after the phenol (0 5 per cent by volume) had been added Pfeiler[14] showed that the causative agent of swine erysipelas was destroyed in 2 weeks by heat generated within piled manure He mentioned the manure must be in piles of not less than a cubic yard in volume and the feces must be mixed with litter in a proportion of 2 to 3 In addition, moisture must be present, and the heat of fermentation should not be less than 140° F Pieces of infected meat 6 inches thick required 2½ hours of cooking before it was sterilized The erysipelas organisms are quite resistant to salting and pickling and to smoking Pieces of meat and bacon may contain the organism either after pickling for 170 days or after 30 days in a mixture of salt and potassium nitrate, according to Hutyra *et al* (1938) In addition, the organisms have been found in a ham 3 months after smoking From a laboratory standpoint *Ery rhusiopathiae* conveniently lends itself to preservation by lyophilization

Under natural conditions, it has been noted that the organism remained alive for 12 days in direct sunlight, 4 months in putrified flesh and 9 months in a buried carcass Glabashki[15] observed the organism to be alive for 3–4 months in pig and mouse carcasses buried 5–7 feet deep, for 5 months in carcasses left to decay on the surface, and for at least 10 months in carcasses kept under refrigeration Hettche (1937) isolated the organism from city sewage containing drainage from abattoirs and stables, and noted that it was not subject to the time of the year He also observed that the organism remained alive much longer in sewage and aquarium water than in tap water The nature of the terrain and soil appears to be associated with

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11 Cited by Hutyra *et al*, 1938

12 Cited by Tenbroeck 1920

13 Cited by Friedberger and Frohner 1908

14 Cited by Van Es 1932 Animal Hygiene Wiley and Sons New York, p 249

15 Cited in *The Veterinary Bulletin* 1956 26 119

Beagle[9]), the accidental access to tankage or to a field of corn, the feeding of new corn, or after the turning of pigs into new pasture In connection with temperature, Brill (1955) found a distinct correlation between the number of cases of erysipelas and the rise in soil temperature Stryszak (1955) did not observe any marked correlation between weather and rain and the occurrence of the disease Further, under experimental conditions the temperature and moisture, as well as low barometric pressure, did not influence the infection when induced orally

The experimental findings of Dubos et al (1955), Smith and Dubos (1956), and Schaedler and Dubos (1956), although working with mice under artificial conditions, showed that susceptibility to infection can be produced by varied non specific procedures A comment by Dubos et al (1955) is worth noting "the state of susceptibility could change from day to day, indeed hour to hour, in response to all stimuli physical, chemical, physiological or emotional — which constituted the total environment ' It can be appreciated that such basic information tends to give credence to many field observations attempting to explain the occurrence of erysipelas on the farm

Sources of Infection

When the sources of swine erysipelas infection are considered, it is necessary to think in terms of many possibilities and not a single entity The organism may be present in the soil as a saprophyte, living on dead or decaying organic matter Soil contamination may have resulted from the improper disposal of dead pigs, and from the manure and urine of affected pigs and rodents The droppings of birds may also be a source of contamination Drinking water and feed, like the soil, may in similar instances become a source of infection

The probability of normal appearing swine acting as carriers and disseminators

of infection has been recognized for many years Bauermeister, Olt, Van Velzen, and Pitt in 1901-7 demonstrated the presence of the organism in tonsils and/or ileocecal valve of normal swine [10] Dale (1937), Geisler (1953), Connell and Langford (1953), Hartwigk and Barnik (1954), Wellmann (1954b), Anusz (1955), and Szynkiewicz (1955) also recovered the organism from the tonsils of apparently healthy pigs Further, Spears (1954) isolated the organism from the femoral red marrow samples of slaughter pigs Connell and Langford, as well as Wellmann, found the isolated organisms to be pathogenic for mice In this connection, Wellmann also observed it was pathogenic for mice whether it was isolated from pigs that had been artificially infected and later shown to be immune or from pigs in herds in which erysipelas had not been observed for many years Connell and Langford, as well as Anusz, noted there was a higher incidence of carriers during the warmer weather Hays and Harrington (1934) described a situation wherein a sow yielded the organism from the tonsils and spleen 7 months after a known attack of erysipelas With relation to vaccination, Hartwigk and Barnik, following an examination of the tonsils and ileocecal crypts of slaughter hogs, could see no apparent differences in animals that had been vaccinated with adsorbate bacterin, with culture and serum, or with serum alone Anusz found the number of carriers in areas where only vaccination with Stuub's vaccine (avirulent culture) was practiced to be almost the same as in areas where the culture-serum method was used Although the evidence points to the carrier animal as a significant source of infection, the relationship has not been sufficiently demonstrated

Mode of Entrance

It has been accepted that the erysipelas organisms gain entry into the body through ingestion of contaminated feed and water Cornevin and Kit, as well as Solleder,

*Cited by R D Shuman and H W Schoening 1 Recapitulation of the swine erysipelas problem Jour Amer Vet Med Assn. 123 301, 1953

**Cited by Tenbroeck, 1920.

requirements for swine prepared by the sub committee on swine nutrition Nat Res Coun
cil, Wash , D C., p 11

BREED, F 1933 Recognition of the most important infectious diseases of swine in the United
States Jour Amer Vet Med Assn 83 656

——— 1937 Swine erysipelas its distribution increasing importance and control Proc U S
Livestock San Assn p 341

——— 1943 Erysipelothrix rhusiopathiae and Pasteurella avicida in chickens Vet Med 38 430

BREED, R. S., MURRAY, E. G. D., and HITCHENS P A 1948 Bergey s Manual of Determinative
Bacteriology, 6th ed The Williams and Wilkins Co Baltimore p 410

BRILL, J 1955 Patogeneza różycy świn Rocz Nauk Rolniaz i Lesnych 66 469

BRUDNJAK, Z., AND KRALJ, M 1955 Znacenje hemokulture u dijagnostici svinjskog vrbanca Vet
Arhiv 25 206

BYRNE, J L., CONNELL, R FRANK J F AND MOYNIHAN, I W 1952 Studies of swine erysipelas
II Cultural characteristics and virulence of strains of Erysipelothrix rhusiopathiae isolated
in different regions of Canada Canad Jour Comp Med 16 129

CALLAWAY, H P., CLARK, R S., PRICE L W and VEZEY S A 1955 Field use of an adsorbed
swine erysipelas bacterin Vet Med 50 39

COLLIER, J R 1951 A survey of beta hemolytic streptococci from swine Proc Amer Vet Med
Assn , p 169

COLLINS, D H., AND GOLDIE, W 1940 Observations on polyarthritis and on experimental Erysipe
lothrix infection of swine Jour Path and Bact 50 323

CONNELL, R 1954 Erysipelothrix rhusiopathiae infection in a northern chipmunk Eutamias
minimus borealis Canad Jour Comp Med 18 22

———, AND LANGFORD, E V 1953 Studies on swine erysipelas V Presence of Erysipelothrix
rhusiopathiae in apparently healthy pigs Canad Jour Comp Med 17 448

———, MOYNIHAN, I W AND FRANK J F 1952 Studies on swine erysipelas I Literature review
and survey of Erysipelothrix rhusiopathiae infection in Canada Canad Jour Comp Med
16 101

COOPER M S PERSONEUS G HARVEY M J AND PERCIVAL R C 1954 Laboratory studies on
erysipelas I Immunization against swine erysipelas and susceptibility of swine to chal
lenge Amer Jour Vet Res 15 591

CREECH, G T 1921 The bacillus of swine erysipelas isolated from urticarial lesions of swine in
the United States Jour Amer Vet Med Assn 109 139

DALE, C N 1937 Rep of Chief Bur Anim Ind U.S.D.A Washington D C p 45

——— 1940 Studies on phenol tolerance of Erysipelothrix rhusiopathiae Monatsh f Vet
Med 1 7

DEDIE, K 1949 Die saureloslichen Antigene von Erysipelothrix rhusiopathiae Monatsh f Vet
Med 1 7

DELPY, L P , Scientific Director L Institut Mérieux à Marcy L'Étoile Rhone France 1955 Per
sonal communication

——— AND HARS E 1953 Observations sur le mode d action des vaccins tués vaccin solubilisé
(Immunigène) contre le rouget du porc Bull Acad vét France 26 539

DEMNITZ, A AND DRAEGER K 1950 Active immunization against swine erysipelas with special
consideration of the erysipelas adsorbate vaccine Veterinarmed Nachricht 2 25

DINTER, Z 1948 Über den haemagglutinationshemmungstest beim Rotlauf Berl Munch
tierarztl Wschr 10 113

——— AND BAKOS, K 1948 Weitere Untersuchungen uber die hamagglutinative Aktivitat des
Rotlaufbacteriums (Bact rhusiopathiae suis) Zeitschr f Hyg 129 263

DOYLE, L P 1949 Rheumatoid disease in swine Proc U S Livestock San Assn p 151

DOYLE T M 1947 The susceptibility of swine fever immune pigs to intracerebral inoculation
with E rhusiopathiae Vet. Jour 103 11

DUBOS R J SMITH, M J AND SCHAEDLER, R W 1955 Metabolic disturbances and infection
Proc Roy. Soc. Med 48 911

EARLE I P AND STEVENSON J W 1952 Rep of Chief Bur Anim Ind U S D A Washington
D C p 33

EVANS W M AND NAROTSKY, S 1954 Two field cases of Erysipelothrix rhusiopathiae infection
in chickens Cornell Vet 44 32

EWALD F W 1955 Über die Dissoziation von Erysipelothrix rhusiopathiae II Über die Veran
derungen der Antigenstruktur im Verlauf der Dissoziation bei Rotlaufbakterien Monatsh
Tierheilk 7 109

FECHNER J 1954–55 Experimentelle Untersuchungen an Schweinen über das Wesen der Rot
laufschutzimpfung unter besonderer Berucksichtigung der Fehlerguellen Wiss Zeitschr
Humbolt Univ Berlin IV 181

FORTNER J 1947 Zwei verschiedene Wege zum Schutze der Schweine gegen den Rotlauf Berl
Munch tierarztl Wschr 12 141

——— 1953 Über den Einfluss der Vererbung bei Rinderleukose und Schweinerotlauf Inter
nat Vet Cong Proc 15 (2) 887

——— AND DINTER Z 1944 Ist das Rotlaufbakterium der alleinige Erreger des Schweinerot
laufs? Zeitschr f Infektionskrankh 60 157

the presence of the organism Lydtin[16] stated that the disease became prevalent mainly in valleys and low lying plains which have slow flowing streams, and in heavy, damp clay soil Hesse[17] observed that the viability of the organism varied in various types of soil, but that soil rich in lime and humus was particularly favorable Harrington (1933) could see no marked preference for soil as the disease occurred on both heavy and sandy soil, however, he mentioned the more serious occurrences were in an area of heavier, gumbo clay soils

Disinfectants

Ery rhusiopathiae is easily killed by commonly used disinfectants, although it must be remembered that the presence of organic matter interferes with the action of most germicidal agents According to Van Es (1932), 5 per cent solutions of phenol and saponified cresols kill the organisms in about 15 minutes The organisms are also killed rapidly when exposed to a 1 per cent solution of either hot lye or sodium hypochlorite

Control

Preventive measures against swine erysipelas, other than through the use of biologics, follow well established principles of disease control Animals should be raised according to prescribed animal husbandry practices relative to housing condition of lots and pastures and management Sanitation is essential, for conditions such as

manure accumulations, debris, and areas of standing water contribute to the maintenance of a potential reservoir of disease The animals should be observed regularly for deviations from their usual attitude Before animal replacements are made, the health and conditions on the premise where they are to be acquired should be inspected Newly purchased animals should be isolated for at least 30 days

When erysipelas does appear, treatment of the sick animals should be instituted early, and those with normal temperatures and appetites moved to an area not recently used, yet where they can be readily observed Dead animals must be buried deeply after being covered first with lime Following the cessation of the outbreak, walls and floors should be thoroughly scraped, scrubbed, and disinfected A hot 1 to 2 per cent lye solution is satisfactory for this purpose It is advisable to eliminate from the herd pigs showing obvious manifestations of chronic erysipelas The lot or pasture in which the infection appeared can be renovated by removing any debris establishing drainage where necessary, and depending on the location, either leaving idle or planting with a forage crop The previously affected areas can be repopulated after several months

Eradication

When one considers the widespread distribution of erysipelas, its association with a wide variety of animals, its apparent ease of adaptiveness to either saprophytic or parasitic existence, and the absence of reliable means of detection, the eradication of this disease seems very remote

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[16] Cited by Friedberger and Frohner 1908
[17] Cited by Van Es and McGrath 1912

REFERENCES

AITKEN, W A 1912 Terramilin in swine erysipelas No Amer Vet 30 2
———— 1950 Acute swine erysipelas. Jour Amer Vet Med Ass 116 41
ANUSZ Z 1965 Rola nosicielstwa włoskowca różycy w patogenezie trzody chlewnej Rocz Nauk Rolniar i Lesnych 66 513
ATKINSON N 1941 Study of some Australian strains of Erysipelothrix Australian Jour Exper Biol and Med Sci 19 45
BARBER, M A 1939 A comparative study of Listerella and Erysipelothrix Jour Path and Bact 48 11
BRADLETTE, F R AND HUDSON, C B 1956 An outbreak of acute swine erysipelas infection in turkeys. Jour Amer Vet Med Ass 41 475
BITSON W M, CRAMPTON L W, CUNHA, T J, ELLIS N R, AND LUECKE, R W 1953 Nutrient

animales Reproduction experimentale d arthritis à bacilles du rouget chez le lapin Doct Thesis Vigot Frères Paris

JULIANELLE, L. A 1911 The identification of *Erysipelothrix* and its relation to *Listerella* Jour Bact 42 385

KARLSON, A G, AND MERCHANT, I A 1911 The cultural and biochemic properties of *Erysipelothrix rhusiopathiae* Jour Amer Vet Med Assn 2 5

KELSER R A AND SCHOEINC, H W 1913 Manual of Veterinary Bacteriology 4th ed The Williams and Wilkins Co, Baltimore p 385

KERNKAMP, H C. H 1941 Endocarditis in swine due to *Erysipelothrix rhusiopathiae* and to streptococci Jour Amer Vet Med Assn 48 132

KLAUDER J V 1938 Erysipeloid as an occupational disease Jour Amer Med Assn 111 1345

———, AND RULE, A M 1944 Sulfonamide compounds in treatment of *Erysipelothrix rhusiopathiae* infections Arch Derm and Syph 49 27

KÖBE, K 1913 Die Aetiologie der infektiösen Gastroenteritis des Schweines und ihre Beziehungen zum Rotlauf Zentralbl f Bakt I Abt Orig 149 401

KONDO, S, AND SUGIMURA, K 1934 Experimental studies on swine erysipelas bacillus found in fishes Jour Jap Soc. Vet Sci 14 111

——— AND ——— 1935 II The pathogenicity and immunizing property for swine of avirulent swine erysipelas bacilli obtained with trypaflavine Jour Jap Soc Vet Sci 14 338

——— YAMADA, S AND SUGIMURA K 1932 Experimental studies regarding living swine erysipelas vaccine I The pathogenicity and antigenic property of swine erysipelas bacilli treated with acridin derivative Jour Jap Soc. Vet Sci 11 148

KONST, H 1945 Chemotherapy of swine erysipelas trials using sulfanilamide sulfapyridine and sulfathiazole in experimental infection of mice Canad Jour Comp Med 9 135

LENTNER, M S 1940 Swine erysipelas control is the corn belt problem Iowa Vet 2 5

LEVINE, N D 1952 *In* Diseases of Poultry Editors H E Biester and L. H Schwarte 3rd ed Iowa State College Press Ames Iowa p 414

MAAS, A 1953 Die Riemser Rotlaufadsorbatvakzine nach Traub in der Praxis 1947 bis 1951 Monatssh f Vet Med, p 1

McNUTT, S H 1934 Swine erysipelas Vet Sci News 8 1

———, LEITH, T S, AND UNDERBJERG, C K 1948 An active agent isolated from arthritis of a pig Amer Jour Vet Res 6 97

MARSH, H 1931 The bacillus of swine erysipelas associated with arthritis in lambs Jour Amer Vet Med Assn 78 57

——— 1933 The serological identity of strains of *Erysipelothrix rhusiopathiae* of ovine and porcine origin Jour Amer Vet Med Assn 82 584

MICHALKA J 1939 Überempfindlichkeit und Immunität bei Schweinerotlauf und Schweinpest und ihre Auswirkungen auf die impfungen Wien tierarztl Monatschr 26 1

MOORE, V A 1892 Mouse septicemia bacilli in a pig's spleen with some observations on their pathogenic properties Jour Comp Med and Vet Arch 13 333

MOROZZI, A. AND CIURNELLI, M 1955 Sul potere patogeno di due ceppi di *Erysipelothrix rhusiopathiae* resi penicillino resistenti Vet Ital 6 1191

MORRILL, C C 1939 Erysipeloid occurrence among veterinary students Jour Infect Dis 55 322

——— 1945 Swine erysipelas Proc U S Livestock San Assn p 92

MOULTON, J E RHODE E R AND WHEAT J D 1953 Erysipelatous arthritis in calves Jour Amer Vet Med Assn 123 335

MOYNIHAN, I W AND STOVELL, P L 1954 The sensitivity of *Erysipelothrix rhusiopathiae* to antibiotics and its relation to chemotherapy Proc. Amer Vet Med Assn p 327

MULLER F 1954 Die Rotlaufdiagnose bei der bakteriologischen Fleischuntersuchung Berl Munch tierarztl Wschr 67 189

MULLER, F L 1955 Vergleichende Praxisuntersuchungen zur Behandlung des Rotlauf Monatsh f Vet Med 10 489

MUNCE, T W 1940 Unknown factors complicate swine erysipelas problem Iowa Vet 11 21

——— 1942 Swine erysipelas No Amer Vet 23 161

MURASE, N AND SHIMIZU, F 1953 Studies on swine erysipelas I Relations between infection and immunity Govt Exp Sta for Anim Hyg Tokyo Japan Exp Rep 27 185

NISHIMURA, Y SATO U AND WATANABE M 1954 Experimental infection of pigs with *Erysipelothrix rhusiopathiae* Jap Jour Vet Sci 16 175

NOWAK B 1952 A contribution to the biology of the Staubs strain of *Erysipelothrix rhusiopathiae* Ann Univ M Curie Sklodowska Lublin Polon 7 149

OSTEEN O L 1941 Rep Chief Bur Anim Ind Washington D C P 58

——— 1942 Rep Chief Bur Anim Ind Washington D C p 27

PARKER, J W LOCKHART, AND RAY, J D 1924 Porcine erysipelas Jour Amer Vet Med Assn 64 451

PARNAS J, LOCKIEWICZ, Z AND POZNANSKA Z 1951 Studies on the increase of virulence of Staubs strain and haemagglutination with *Erysipelothrix rhusiopathiae* Ann Univ M Curie Sklodowska Lublin Polon 9 41

——— Curie Sklodowska Lublin Polon Jour Amer Vet Med Assn 10s 10

PETERMAN, J E 1944a Diagnostic chart of swine erysipelas Jour Amer Vet Med

FRIEDBERGER AND FROHNER 1908 Veterinary Pathology Hayes 6th English ed W T Keener and Co, Chicago Vol 2 p 221

GEISLER S 1953 Untersuchungen uber das Vorkommen von Rotlaufbakterien in den Tonsillen gesunder Schlachtschweine Zentralbl f Bakt I Abt Orig 159 335

GELENCZEI, E, AND RAPPAY, D 1955 Hyperimmunization von Pferden mit Rotlaufbazillenstam men des A und B Type Acta Vet Hung 5 1

GILTNER, L T 1922 A fatal disease of young pigs apparently caused by the bacillus of swine erysipelas Jour Amer Vet Med Assn 61 540

GLEDHILL, A W 1945 The antigenic structure of Erysipelothrix Jour Path and Bact 57 179

———— 1947 Some properties of a thermolabile antigen of *Erysipelothrix rhusiopathiae* Jour Gen Microbiol I 211

GODGLUCK, G AND WELLMANN, G 1953 Rotlaufbakterien in der Haut und in Blut bei experi mentell mit Rotlauf infizierten Schweinen Deutsch tierarztl Wschr 60 537

GORHAM, J R 1949 An attempt to infect mink and fox with *Erysipelothrix rhusiopathiae* Vet Med 44 136

GOUGE, H E BOLTON, R, AND ALSON, M C 1956b Laboratory studies on erysipelas III Duration of immunity in pigs vaccinated with adsorbed bacterin, and with serum and culture Amer Jour Vet Res 17 135

———— ———— AND BROWN, R 1956a Laboratory studies on erysipelas II Use of various cultures in production of infection in pigs by skin scarification Amer Jour Vet Res 17 132

GRAHAM, R, LEVINE, N D, AND HESTER, H R 1939 *Erysipelothrix rhusiopathiae* associated with a fatal disease in ducks Jour Amer Vet Assn 95 211

GRAY, C W, AND NORDEN, C J 1955 Erysipelas vaccine avirulent—a new agent for erysipelas control Jour Amer Vet Med Assn 127 506

GREY, C J 1944 State Experiment Stations Division, A R S, U S D A Washington D C formerly with the B A I U S D A Personal communication, 1956

———— 1947a Personal communication, 1956

———— 1947b Effects of penicillin on *Erysipelothrix rhusiopathiae* and on mice infected with that organism Vet Med 42 74

———— OSTEEN O L AND SCHOENING H W 1941 Swine erysipelas the agglutination test for its diagnosis and a report on a study of arthritis in swine Amer Jour Vet Res 2 74

GWATKIN, R 1940 Swine erysipelas Canad Jour Comp Med 4 236

HARRINGTON, C F 1938 Field observations on swine erysipelas in swine herds Jour Amer Vet Med Assn 82 492

HARS, E AND DELPY, L P 1953 Inoculation de E rhusiopathiae par voie intradermique Ap plication au titrage des vaccins et sérums contre le rouget du porc Bull Acad vét France. 26 267

HARTSOUGH, G R 1945 Isolation of Erysipelothrix rhusiopathiae from farm raised mink Jour Amer Vet Med Assn 107 242

HARTWIGER, H, AND BARNIK K F 1954 Ein Beitrag zur Frage der Schaffung von Rotlaufbakter reintrager durch die Simultanimpfung Deutsch tierarztl Wschr 61 241

HAYS, C H, AND HARRINGTON, C F 1934 Swine erysipelas as a herd involvement Vet Alum Quart Ohio 22 95

HEILMAN, F R, AND HERRELL W E 1944 Penicillin in the treatment of experimental infections due to *Erysipelothrix rhusiopathiae* Proc. Staff Meet Mayo Clinic 19 340

HETTCHE, H O 1937 Zur Atiologie der Rotlaufinfektion Archiv f Hyg u Bakt 119 178

HOLM, G C, GRIFFITH, R L, JR, AND BEESON, W M 1942 Chronic swine erysipelas Vet Med 37 123

HRUSKA, K 1952 Srovnávaci studie o cervence vepru II Aktivni imunita proti cervence vepru a novi cesty pripravy vakein Ceskoslov Akad Zemedel Sborn 25 541

HUBBARD, E D 1952 Hemocultures as a diagnostic aid in swine erysipelas Jour Amer Vet Med Assn 120 291

HUGHES, D L 1953 Discussion of swine erysipelas Vet Rec 65 713

———— 1955 Arthritis in pigs III The experimental disease induced by Erysipelothrix rhusiopathiae Brit Vet Jour III 183

HUTNER, S H 1942 Some growth requirements of *Erysipelothrix* and *Listerella* Jour Bact 43 629

HUTYRA, J, MAREK, J, AND MANNINGER, R. 1938 Special Pathology and Therapeutics of the Diseases of Domestic Animals, 4th English ed Alexander Eger, Chicago Vol 1, p 76

JANOWSKI, H 1951 Badania nad podniesieniem wartósci uodporniających nierzadliwej kultury różycowej według Stauba 1 Wpływ zwickszonej dawki (5 ml) szczepionki na adporność Rocz Nauk Rolnicz 1 Lesnych 67 233

JANSEN J, VAN DORSSEN, C H, AND FREDERIK, A H 1956 Deformerende arthritis en peri arthritus door Erysipelothrix rhusiopathiae — infectie bij jonge varkens Tijdschr v Dier geneesk 81 63

JARVIS, M K 1956 Director of Laboratories, Corn State Laboratories, Inc., Omaha, Nebr Per sonal communication

JEAN, E 1934 Pouvoir pathogène et localisation du bacille du rouget dans les différentes espèces

———, ——, AND ——— 1955b II The effects of hormonal therapy on advanced chronic polyarthritis experimentally induced by *Erysipelothrix* infections Amer Jour Vet Res 16 367.

———, ——, AND ——— 1956 Swine erysipelas II Prophylactic effect on a commercially prepared bacterin in swine Jour Amer Vet Med Assn 128 283

SMITH, H C 1955 Advances made in swine practice Vet Med 50 683

SMITH, M J AND DUBOS, R J 1956 The effect of nutritional disturbances on the susceptibility of mice to staphylococcal infections Jour Exper Med 103 109

SMITH, T 1885 2nd Ann Rep Bur Anim Ind Washington D C p 187

——— 1895 12th and 13th Ann Rep Bur Anim Ind Washington D C p 166

SPEARS, H N 1954 Carriers of swine erysipelas Jour Comp Path and Therap 64 152

SPENCER, G R 1954 The pathogenesis of swine erysipelas Proc Amer Vet Med Assn p 132

STAUB, A 1939 Sur la vaccination contre le rouget du porc C R Acad Sci Paris p 775

——— 1940 Nouvelle formule de vaccination contre le rouget du porc Bull Acad vét France 13 103

STILES, G W 1944 Swine erysipelas organisms recovered from a brown rat (*Rattus norvegicus*) Amer Jour Vet Res 5 243

———, AND DAVIS, C L 1931 Swine erysipelas and its economic importance Jour Amer Vet Med Assn 84 895

STRYSZAK, A 1955 Rola czynników środowiskowych w patogenezie różycy Rocz Nauk Rolniaz i Leśnych 66 509

———, AND OYRZANOWSKA, J 1955 Ustalenie drog naturalnego zakazania sie białych myszy włoskowcem różycy z uwzglednieniem wpływu temperatury Rocz Nauk Rolniaz i Leśnych 66 549

SUGIMURA K 1955 Personal communication through Dr M Kobayashi Chief N I H A Tokyo

SWAN L C 1949 Observations on an arthritic condition in swine associated with pellagra Canad Jour Comp Med 13 65

SWITZER W P 1954 A suspected PPLO in Iowa swine Iowa Vet 25 9

SZYNKIEWICZ, Z 1955 Nosicielstwo w włoskowców różycy na migdałkach świn poddanych obojowi Rocz Nauk Rolniaz i Leśnych 66 535

TAYLOR J B 1931 Swine erysipelas Jour Amer Vet Med Assn 79 813

TENBROECK, C 1920 Studies on *Bacillus murisepticus* or the rotlauf bacillus, isolated from swine in the United States Jour Exper Med 32 331

THOMSON, A, AND GLEDHILL A W 1953 The demonstration of the protective value of a swine erysipelas vaccine in pigs Vet Rec 65 40

TIFFANY, L W 1955 A suitable basal medium for evaluation of the fermentative capacity of *Erysipelothrix rhusiopathiae* Amer Jour Vet Res 16 636

TOPLEY, AND WILSON 1946 Principles of Bacteriology and Immunity 3rd ed The Williams and Wilkins Co Baltimore p 395

TRAUB, E 1947 Immunisierung gegen Schweinerotlauf mit konzentrierten Adsorbatimpfstoffen Monatsh f Vet Med 10 165

TRAUTWEIN K 1949 Epidémiologie et prophylaxie du rouget du porc Bull Off int Epiz 32 222

TRAWINSKI A 1949 Preventative vaccination of pigs against swine erysipelas F.A.O Agr Studies, Washington D C 101

USDIN, M, AND BIRKELAND, J M 1949 The production of hyaluronidase by *Erysipelothrix rhusiopathiae* and its possible relationship to polyarthritis in swine Bact Proc Soc. Amer Bact p 75

——— FERGUSON L C AND BIRKELAND J M 1952 Experimental arthritis in swine following multiple injections with *Erysipelothrix rhusiopathiae* Amer Jour Vet Res 13 188

VAN Es L 1932 Animal Hygiene Wiley and Sons New York p 514

——— AND McGRATH C B 1936 Swine erysipelas Nebr Agr Exp Sta Res Bull No 84

——— AND ——— 1942 Swine erysipelas Nebr Agr Exp Sta Res Bull 128

——— OLNEY J F AND BLORE I C 1940 Some factors influencing swine erysipelas prophylaxis Proc U S Livestock San Assn p 34

——— AND ——— 1945 The effects of penicillin on *E rhusiopathiae* infected pigeons Nebr Agr Exp Sta Res Bull 141

WALLER, E F 1938 Swine erysipelas in newborn pigs Jour Amer Vet Med Assn 46 196

WARD A R 1922 The etiology of polyarthritis in swine Jour Amer Vet Med Assn 61 155

WATTS, P S 1950 Studies on *Erysipelothrix rhusiopathiae* Jour Path and Bact 50 355

WAYSON, N E 1927 An epizootic among meadow mice in California caused by the bacillus of mouse septicaemia or of swine erysipelas Pub Health Rep 42 1489

WELLMANN G 1949 Die Übertragung des Schweinerotlaufs durch den Saugakt der gemeinen Stechfliege (*Stomoxys calcitrans*) und ihre epidemiologische Bedeutung Berl Munch tierärztl Wschr p 39

——— 1953 Auftreten von Rotlaufbakterien im Blut von experimentell mit Rotlauf infinierten Schweinen Deutsch tierärztl Wschr 60 366

PETERMAN, J E 1944b Swine erysipelas Vet Med 39 53

———, SCHOENING, H W BEAGLE, A G., AND GREY, C G 1948 The cooperative state and federal project on the control of swine erysipelas through the use of culture and serum Proc 52nd Ann Meet U S Livestock San Assn, p 132

PHILLIPS C E 1956 Swine immunity with erysipelas bacterin So West Vet 9 115

PORTER, J R, AND HALE, W M 1939 Effect of sulfanilamide and sulfapyridine on experimental infections with *Listerella* and *Erysipelothrix* in mice Proc Soc Exper Biol and Med 42 47

PRIER, J E, AND ALBERTS, J O 1950 The effects of aureomycin and of penicillin against *Erysipelothrix rhusiopathiae* in vitro and in vivo Jour Bact 60 139

RADVILA P 1953 Hamagglutination Autolyse und Immunisierungs Vermogen von Schweine rotlaufbazillen Schweiz Arch f Tierheilk 95 33

RAILSBACK, L T 1951 Swine erysipelas Iowa Vet 22 12

——— 1956 Elsworth, Minn Personal communication

RAY, J D 1931 Arthritis in lambs and *Erysipelothrix rhusiopathiae* Jour Amer Vet Med Assn 30 107

RICE, C E BYRNE, J L CONNELL, R, MOYNIHAN I W, AND FRANK J F 1952a Studies on swine erysipelas III Antigenic characteristics of strains of *Erysipelothrix rhusiopathiae* isolated in different areas in Canada Canad Jour Comp Med 16 195

———, CONNELL, R, BYRNE, J L, AND BOULANGER, P 1952b Studies of swine erysipelas. IV Serological diagnosis in swine Canad Jour Comp Med 16 209

ROOTS, E. AND VENSKE W 1952 Serologische und immunogene Eigenschaften der *Erysipelothrix rhusiopathiae* (E muriseptica) I Die Antigenstruktur und die Methoden des Nachweises der Serotypen Berl Munch tierarztl Wschr 65 208

ROWSELL, H C 1955 Studies on the experimental production of swine erysipelas Proc. Amer Vet Med Assn, p 143

SANDSTEDT, H H, AND LEINVERT, E 1944 Erfarenheter av under 1943 utforda ympningar mot rod sjuka hos svin Skand Vet Tdskr 34 129

——— AND SWAHN, O 1947 Ympning av smågrison med avirulent rodsjukeympamne Skand Vet Tdskr 37 85

SATOH, H OHMAYASHI M, AND ONO, T 1953 Histopathological investigations on acute swine erysipelas Vet Res 1 111

SCHAEDLER, R W AND DUBOS, R J 1956 Reversible changes in the susceptibility of mice to bacterial infections Jour Exper Med 104 67

SCHATZ, A AND WAKSMAN, S A 1944 Effect of streptomycin and other antibiotic substances on *M tuberculosis* and related organisms Proc. Soc. Exper Biol and Med 57 244

SCHELLNER H H, AND SCHLEIBHEIM, F S 1949 Beiträge zum Rotlaufproblem Tierärztl Umschau p 29

SCHNEIDER, B H, SPENCER, G R, AND ENSMINGER, M E 1955 Antibiotic feeding for prophylaxis of experimentally produced swine erysipelas Jour Canad Sci 14 1140

SCHOENING H W, AND CREECH, G T 1935 Serological studies of swine erysipela with particular reference to agglutination Jour Agr Res 50 71

——— AND ——— 1936 Swine erysipelas—with particular reference to serological diagnosis. Jour Amer Vet Med Assn 88 310

——— ——— AND GREY, C G 1932 A laboratory test tube and a whole blood rapid agglutination test for the diagnosis of swine erysipelas. No Amer Vet 27 19

———, GOCHENOUR, W S, AND GREY, C G 1938 Studies on *Erysipelothrix rhusiopathiae* with special reference to smooth and rough types of cultures Jour Amer Vet. Med Assn 92 61

———, PETERMAN, J E, AND GREY C G 1940 A study of an outbreak of acute swine erysipelas in one herd Jour Amer Vet Med Assn 46 714

SEIBOLD, H R, AND NEAL, J E 1956 Erysipelothrix septicemia in the porpoise Jour Amer Vet Med Assn 128 537

SHUMAN R D 1950 Rep Chief Bur Anim Ind, Washington, D C., p 46

——— 1951 Swine erysipelas induced by skin scarification Proc. Amer Vet Med Assn, p 153

——— 1953a Experimental evaluation of culture and serum vaccination for the control of swine erysipelas II Baby pig vaccination Jour Amer Vet Med Assn 123 504

——— 1953b III Weaning age vaccination Jour Amer Vet. Med Assn 123 507

——— 1953c IV Gilts vaccinated with culture and serum before breeding, and its immunizing effect on their offspring Jour Amer Vet Med Assn 123 434

——— 1954 Experimental evaluation of swine erysipelas adsorbate bacterin Jour Amer Vet Med Assn 124 362.

——— AND EARL, F L 1954 V Vaccination of weanling pigs using a change in the dosage relationship of culture and serum Jour Amer Vet Med Assn 125 506

——— AND LEE, A. M. 1950 The susceptibility of hamsters to *Erysipelothrix rhusiopathiae* Jour Bact 60 677

SIKES, D, NEHR, G M, AND DOYLE, L P 1955a Studies on arthritis in swine I Experimental erysipelas and chronic arthritis in swine Amer Jour Vet Res. 16 319

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Pasteurellosis

Pasteurellosis is an infectious disease due to a specific microorganism, *Pasteurella multocida* Pasteurellosis rarely occurs as a primary disease but develops as a disease secondary to forces operating concurrently which tend to devitalize the pig It occurs in two forms (*a*) as a subacute or chronic disease of the lungs, (*b*) as an acute septicemic disease

Swine plague and hemorrhagic septicemia are names that, for many years, have been used to describe this disease It was called swine plague long before emergence of the more enlightened knowledge of the causes of disease Plague was a term used to denote pestilences and enzootics Since troublesome diseases existed among swine, it seems quite natural that the name swine plague was used to designate the condition that was considered a specific disease entity The name hemorrhagic septicemia came into use after more was learned about some of the bacteriological and pathological characteristics of the disease The punctiform and ecchymotic hemorrhages observed at the necropsy of pigs dead from this disease, plus the isolation of the organism in pure culture from the blood and other tissues, were accepted as valid reasons for selecting hemorrhagic septicemia as an appropriate name Merchant and Packer (1956) pointed out that in 1900 Lignieres proposed the term pasteurellosis to designate the diseases due to organisms of the genus Pasteurella They favored the use of pasteurellosis as the specific name for this disease and point out that it is finding its way back into the literature

Pasteurellosis may occur in a herd as a sporadic disease in which one or a small number of the pigs of the herd are affected or as an enzootic disease affecting a relatively large proportion of the swine in the herd Swine of all ages are susceptible but the disease occurs most frequently in shoats from 50 to 150 pounds in weight The losses from pasteurellosis vary greatly and to a considerable extent depend upon the virulence of the infecting organism and/or the conditions and circumstances under which it prevails

All farm animals, including cats and dogs, are susceptible to infection with *P multocida* In this connection it is important to keep in mind that although the cultural and biochemical characteristics of *P multocida* isolated from swine affected with pasteurellosis are similar to the cultural and biochemical characteristics of the organisms in the genus Pasteurella obtained from cattle, sheep, or chickens affected with the same disease, the disease in swine seldom spreads to members of another class of animals This is true even when affected swine come in close contact with cattle, sheep, or chickens in a stable,

WELLMANN, G   1954a Rotlaufinfektionsversuche an wilden Mausen Sperlingen Huhnern und
     Puten Tierarztl Umschau 9 269
———— 1954b Vorkommen und Virulenz von Rotlaufbakterien in nicht an akutem Rotlauf
     erkrankten Schweinen Deutsch tierarztl Wschr 61 357
———— 1955a Die subklinische Rotlaufinfektion und ihre Bedeutung fur die Epidemiologie des
     Schweinerotlaufs Zentralbl f Bakt, I Abt Orig, 162 265
———— 1955b Die Übertragung der Schweinerotlaufinfektion durch die Stubenfliege (*Musca
     domestica*) Zentralbl f Bakt I Abt Orig, 162 261
———— 1956 Bundesgesundheitsamt Max von Pettenkoferinstitut, Berlin Dahlem Personal
     communication
WIEBNER W   1952 Die kombinierte Penicillin Formolvakzine Behandlung des Schweinerotlaufs.
     Wien tierarztl Monatschr 39 352
WIDIR R. W   1952 Die wissenschaftlichen Grundlagen der akuten Immunisierung gegen den
     Schweinerotlauf mit dem schwedischen avirulant Impfstoff Monatsh f prakt Tierheilk
     4 145
———— 1955 Statens Veterinarmedicinska Anstalt Stockholm Personal communication
———— AND EILERS T   1953 Vergleichende Untersuchungen über die Rotlaufimmunitat der
     Schweine nach Impfung mit dem schwedischen avirulenten Rotlaufimpfstoff und Rotlauf
     adsorbatimpfstoff 15th Internat Vet Cong Proc. 1 (1) 49
WIX P AND WOODBINE, M 1955 Microbiological aspects of the swine erysipelas organism
     Erysipelothrix rhusiopathiae Brit. Vet Jour 111 432
WOODBINE M 1947 Chemotherapy of *Erysipelothrix rhusiopathiae* infection in mice with strep
     tomycin Vet. Jour 103 149
———— 1950 *Erysipelothrix rhusiopathiae*, bacteriology and chemotherapy Bact Rev 14 161

infestations and that harbor *P multocida* sometimes develop pasteurellosis

CLINICAL SIGNS

The clinical signs of the pectoral form of pasteurellosis are similar to those characteristic of most inflammatory diseases of the lungs due to an infectious agent Cough, dyspnea, fever, anorexia, and general physical weakness are important signs in pneumonia from any cause

With the engorgement of the lungs and the accumulation of the inflammatory exudates in the bronchioles and alveoli, the ventilation function of the lungs is markedly reduced and breathing becomes difficult A labored, "thumpy," abdominal type of breathing is quite common in advanced stages of this disease When the breathing is difficult, the pig sometimes assumes a sitting position, extending its head and respiring through its mouth In the early stages of the consolidation phase of the pneumonia, crepitant sounds may be heard when auscultating the thorax When the sounds become more characteristic of gurgling and bubbling, it is an indication that the exudates are beginning to break down and move with the air in the bronchi The cough, a very common symptom in lung disease, is usually dry and hacking in the early stages and more moist later in the course of the disease A muco purulent discharge from the snout occurs in many of the cases The body temperature is elevated to 105°–106° F If water is available and the pig is not too weak to move about, it will drink small amounts at frequent intervals

The clinical course of subacute cases usually extends over a period of 5 to 8 days and then ends in death In the more chronic cases the clinical course covers a much longer period Infected animals may live from 3 to 5 weeks, with 30 to 40 per cent recovering In these long, drawn-out cases, emaciation with general weakness is common

The septicemic form is marked by a sudden onset, extreme physical weakness, a very incoordinate gait, and death in a high proportion of the cases The affected pigs manifest a depressed general attitude and have no desire to take feed The body temperature is elevated to 105°–106° F The clinical course is short and usually ends in death in 10 to 36 hours

When pasteurellosis occurs as a complication to other infectious or metabolic diseases it often supercedes the first or primary disease to produce a syndrome in which the pulmonary disorder becomes the number one factor of distress

PATHOLOGICAL CHANGES

In the pectoral form, the lungs and adnexa are the principal sites of tissue alteration The extent of the lesions varies with the virulence of the organism and the duration of the disease In early stages patchy and scattered areas of consolidation in the different lobes are commonly observed These are lobules in which the inflammatory exudate is accumulating The color of the affected area may be reddish or greyish, depending somewhat upon the stage of the inflammatory process With a spreading of the inflammation by direct extension or by metastasis, large portions of the lungs, or even the entire lungs in some cases, become involved The lungs are distended, quite firm, and do not collapse when the thorax is opened The cut surface usually appears mottled, with the colored areas varying from reddish through yellowish to grayish and representing different stages in the inflammatory process Frequently, varying amounts of edema also are present in the interlobular spaces Small necrotic areas may be seen on the cut surface and the mucous membranes of the larger bronchi The trachea often is reddened and swollen Many times one finds petechiae on the pericardium and more often on the endocardium The mediastinal lymph nodes are swollen and hemorrhagic Very often an exudative pleuritis that covers practically the entire surface of the lung will be present The visceral pleura is reddened, thickened and beadlike in appearance

The necropsy findings in the septicemic form are characterized by petechiae and ecchymoses in the serous and mucous membranes

barnyard, or pasture Rabbits and mice are highly susceptible to infection with *P multocida*

This disease has a worldwide distribution From time to time during the past 25 years, reports of this disease have appeared in the veterinary literature from different parts of the world Some workers consider it a specific and primary disease in swine, but others consider it important mainly as a secondary disease The latter view is emphasized in this chapter

ETIOLOGY

The role of *P multocida* as a primary cause of pasteurellosis in swine has been controversial for many years There is general agreement that it acts with other stresses to produce the disease described here Most veterinarians, who have had some experience with the relationship of *P multocida* to the development and progress of the disease, hold that this organism is capable of producing disease only in animals whose resistance has been lowered *considerably* At the same time, Fox and Burkhart (1947), Kelser and Schoening (1948), and Merchant and Packer (1956) state that the virulence of the organism may increase during the course of an outbreak and become the definitive and primary cause of the disease We have had contact with outbreaks of the septicemic form of pasteurellosis which would support this view

The relationship of *P multocida* to the onset and progress of this disease in swine is aptly stated by Hagan and Bruner (1951), Presumably under conditions of lowered host vitality, organisms that are already carried on the respiratory mucous membranes are unleashed and assume a pathogenicity which they did not possess formerly " It is not rare or unusual to isolate this organism in pure culture from the lungs of swine that did not manifest gross tissue alteration Spray (1922) made a study of the bacterial flora of normal and diseased lungs of swine slaughtered at a meat packing institution in Chicago He found six per cent of 100 normal lungs examined harbored *Bacillus suisepticus* (P.

multocida) In diseased lungs, this organism was isolated in pure culture from 44 per cent of 314 swine

P multocida, according to Bergey's Manual (1948), are short ellipsoidal rods They are nonmotile, Gram negative, and have a bipolar staining characteristic They are aerobic or facultative anaerobic and will grow on ordinary culture media at an optimum temperature of 37° C

The debilitating and predisposing factors contributing to the onset and development of pasteurellosis concern such things as the environmental temperature and barometric conditions, shelters or stabling facilities, the state of nutrition from a quantitative and qualitative standpoint, and the presence of other infectious and/or parasitic diseases Environmental temperatures that drop from comparatively warm to decidedly cool within a short period of time — 3 to 6 hours — usually have an effect on the metabolic activity of the animal which tends to lower its vitality and resistance When, in addition, the atmosphere is humid, the stress due to climatic influences is increased Such conditions are conducive to the development of pasteurellosis in pigs harboring the specific infectious agent and in others that might 'pick up" the agent by contact Events that sometimes occur during transportation or movement of swine from one environment to another often contribute to the onset of this disease Malnutrition bears a certain relationship to susceptibility, so that swine receiving an inadequate ration have a lowered resistance and would be more susceptible to the bacterial agent if it were present.

The simultaneous existence of pasteurellosis and certain other infectious diseases is not uncommon The pectoral form of this disease is frequently a complication of hog cholera that may be due to a virus of low virulence or of cholera in which the clinical course is of comparatively long duration Pasteurellosis is a terminal complication in many cases of swine influenza and of chronic salmonellosis Swine that are devitalized as a result of heavy worm

recipient a state of passive immunity This can be employed as a prophylactic measure where a temporary immune state is desired immediately.

Good care and nursing are important adjuncts to any course of treatment. Clean water and nutritious feed should be available at all times. It is highly desirable and quite important that the sick swine be isolated from the remainder of the herd and that they be placed in clean, dry quarters that are free from drafts

REFERENCES

BERGEY, D H, BREED, R S, MURRAY, E G D, AND HITCHENS A P 1948 A Manual of Determinative Bacteriology, 6th ed Williams and Wilkins Co, Baltimore

FOX, O K, AND BURAHART, R L 1947 Hemorrhagic septicemia in swine controlled with sodium sulfamethazine Vet Med 42 379

HAGAN, W A, AND BRUNER, D W 1951 The Infectious Diseases of Domestic Animals, 2nd ed Comstock Publishing Co, Ithaca N Y

KELSER, R A, AND SCHOENING, H W 1948 Manual of Veterinary Bacteriology, 5th ed Williams and Wilkins Co, Baltimore

LARSEN, C E 1948. Sulfamethazine in the treatment of Pasteurellosis and dysentery in swine Vet Med. 43 231

MERCHANT, I A, AND PACKER, R A 1956 Veterinary Bacteriology and Virology, 5th ed The Iowa State University Press, Ames, Iowa

SPRAY, R S 1922 The bacteria in normal and diseased lungs of swine Jour Infect Dis 31 10

branes of the gastrointestinal tract, the pericardium, endocardium, and sometimes in the skin In many of these cases, the lungs will contain relatively large amounts of edema fluids in the interlobular septal spaces, in the bronchi and alveoli The kidneys are hyperemic and may be petechiated

DIAGNOSIS

The fact that the pig suffers from a disease of the lung is readily appreciated The clinical signs and necropsy findings leave no doubt about this conclusion Diagnosis of the specific cause or at least the significant contributing cause of the pneumonia depends upon laboratory techniques The isolation of *P multocida* in pure or mixed cultures is necessary before a final diagnosis of the case can be made On the other hand, a pasteurellosis diagnosis can be made with a high degree of accuracy by a critical analysis of all available factors pertaining to conditions affecting the herd as a whole Very often, only one or a small number of pigs of the herd are affected Swine influenza, acute salmonellosis, acute swine erysipelas and hog cholera usually affect a larger proportion of the herd by the time the disease has progressed in the herd to the point where veterinary services are employed One also takes into account any or all of the conditions, affecting the pig and/or the herd, which have been discussed as devitalizing agencies that contribute to the occurrence of pasteurellosis If necropsy is possible the rather extensive bilateral pneumonia, showing consolidation and much interlobular edema, suggests pasteurellosis

The septicemic form may be mistaken for peracute hog cholera, especially when dealing with pasteurellosis early in the outbreak The physical signs of the two diseases and even some of the necropsy findings make the differential diagnosis difficult A count of the number of white blood cells per unit volume of blood (cu mm) may be very helpful Counts of 20,000 and more are usually found in pasteurellosis The particular value of a leukocyte count is that it may help to eliminate

the hog cholera (see Chapter 7, Hog Cholera) However, before one can be certain about the diagnosis of pasteurellosis, the specific bacterial cause, *P multocida* should be obtained from the blood and/or other tissues

TREATMENT

The treatment of this disease is not altogether satisfactory, and particularly when the pneumonia has progressed to the stage where it becomes a lobar type On the other hand, early and adequate use of sulfonamides will reduce the death rate from this disease Excellent results were reported by Fox and Burkhart (1947) and by Larsen (1918) on the use of sulfamethazine Fox and Burkhart used the sodium salt of sulfamethazine and administered it subcutaneously at the rate of 15 grains per lb body weight On the second and third day of treatment the dose was reduced to 1 grain per lb According to Larsen a single dose of sulfamethazine given intraperitoneally at rate of 1 grain per lb body weight was very effective

Sulfamerazine, 1 grain per lb the first day and ½ grain per lb every 12 hours for the following 2 to 3 days, can be employed It should be administered by way of mouth If the sodium salt of sulfamerazine is used, it can be injected directly into the blood stream or into the peritoneal cavity Sulfathiazole is another of the sulfa drugs that has a place in the treatment of this disease It is used according to the schedule described for sulfamerazine

Aqueous penicillin in doses of 50,000 units administered intravenously every 24 hours for 2 or 3 days may be used to treat pigs affected with this disease, or it may be more desirable to use a penicillin in oil preparation and inject it into the muscle in doses of 500,000 to 1,000,000 units. Dihydrostreptomycin, 2 mg per lb body weight administered intramuscularly is about equally effective Oxytetracycline, 1 to 2 mg per lb given intramuscularly, is also effective in the treatment of pasteurellosis.

Hemorrhagic septicemia antiserum in doses of 25 ml per 100 lb conveys to the

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Streptococcosis and Colibacillosis

Streptococcosis

Pathogenic streptococci tend to localize in specific tissues although septicemic conditions have been known to occur These localizations tend to produce abscesses (see Chapter 48) However, all localized infections are neither characterized by walled off pyogenic processes nor are they always associated with the production of pus

CENTRAL NERVOUS SYSTEM INFECTION

Most common of the non abscessing streptococcic infections are those affecting the brain and the meninges McNutt and Packer (1943) described several cases of central nervous system infections in which streptococci were isolated There appeared to be no specific strain of streptococci involved but rather a number of strains, none of which appeared to belong to any of the recognized pathogenic species Also, the strains were relatively noninfectious for guinea pigs It is seen in Table 21 1 that not more than two strains conformed to any one set of reactions

The animals reported in these studies were largely under 8 weeks of age, although one group averaged about 90 pounds Marked evidence of encephalitis was encountered, characterized by convulsions and death within 30 to 36 hours. While gross lesions in the brain (or other parts of the body) were not evident, a histological examination of the brain and meninges

revealed perivascular and focal areas of infiltration with inflammatory cells which were chiefly polymorphonuclear leukocytes Streptococci were isolated from the brain and meninges but not from other organs in the body The authors indicated that in two of the three herds in which streptococcic infection occurred, some degree of anemia was observed They further suggested that the bacterial infection was probably the result of secondary infection of an animal in which resistance had been lowered by some other disease process including those caused by filtrable viruses The disease was most common in winter and spring months when resistance was lowest

Streptococcal infection of the central nervous system of baby pigs ranging from 2 to 6 weeks of age was also reported by Field *et al* (1954) The organisms sometimes infected only one pig in one litter but often spread to a number of pigs in many litters Season and management were not considered to be factors

Signs included an early temperature rise and anorexia Pigs buried themselves in bedding swayed when walking and had a weakness of the hind quarters and a stiff legged gait They often assumed a crouch ing position with ears retracted close to the sides of the head Sometimes they were found shaking violently Poor growth and swollen joints often were the chronic ef fects of the infection

The organisms were readily cultured from the brain, less frequently from the viscera and occasionally from the joints They were Gram positive cocci occurring singly or in pairs and less frequently in chains Alpha hemolysis was produced on sheep blood agar and beta hemolysis on horse blood agar Growth in nutrient broth was poor The addition of 10 per cent horse serum improved growth Acid but no gas was produced in trehalose, salicin, inulin, lactose dextrose maltose, levulose, sucrose, and galactose Growth in sorbite, manite raffinose arabinose dulcite, glycerin, in ositol rhamnose and xylose produced no change Some strains produced a partial reduction of 1 5000 methylene blue milk Acid was produced in litmus milk Starch was hydrolyzed The organism did not show a serological relationship to any of the Lancefield groups

The acute lesions produced by the strep tococci of Field et al occurred only in the encephalitic form The volume and pres sure of the cerebrospinal fluid were in creased Vascular congestion of the brain and mild suppurative inflammation of the meninges were observed The exudate at times blocked the ventricles, aqueduct, and central canal of the medulla and cord Histologically the condition was described as an acute fibrino-purulent choriomenin gitis with secondary extension to the sub pial and periventricular tissues with lique faction necrosis Hydroperitoneum oc curred in pigs dying in the acute febrile period

Ray (1945) described an inner ear in fection (otitis media) with streptococci which was characterized by the way the animal carried its head to one side The severity of the reaction was dependent upon the degree of involvement. Death occurred when the infection extended to the meninges and the brain Cultural ex amination of early cases was difficult be cause of the small area and bony enclosure of the parts In the advanced stages the animals showed exaggerated nervous symp toms circled aimlessly, and paddled when too weak to stand

SEPTICEMIC INFECTION

Septicemic infection with streptococci was observed in hog cholera immune sows and pigs by Bryant (1945) Three of 10 sows and about 30 of 80 pigs died Strep tococci were isolated from a variety of tis sue specimens submitted to the laboratory The onset of the disease was sudden with death generally occurring in 12 to 18 hours Less often the course of the disease was extended to 36 or 48 hours Animals be came weak and prostrate Temperatures were elevated Scours, bloody urine, and cutaneous hemorrhages ranging from pe techial to large blotches were the most important signs of the disease Upon necropsy, dark congested lymph nodes with peripheral hemorrhage, petechiation, and ecchymosis of lungs, epiglottis, and tonsils were common Blood in the hilus of the kidney and in the gall bladder sometimes occurred The kidneys, ureters, and blad der were dark and hemorrhagic

Ray (1945) suggested that wound con tamination was one factor predisposing to generalized infection He described the lesions of streptococcic septicemia as those characteristic of hog cholera and indicated that the two diseases often existed in swine as mixed infections Diagnosis was best accomplished by culture Mixed infection can only be detected by culture and by the inoculation of tissue or blood filtrates into hog cholera susceptible and hog cholera immune swine

HEART, JOINT, AND STOMACH LESIONS

Upon necropsy, one of the more easily recognized lesions of chronic streptococcosis is the vegetative endocarditis observed in pigs 12 weeks of age and older The lesion is identical in gross appearance to the lesion produced by *Erysipelothrix rhusio pathiae* Animals showing this lesion lack endurance in physical exertion, and usu ally die suddenly Death is commonly due to coronary or encephalic infarction The mitral, tricuspid, and aortic valves separ ately or as a group are grossly thickened and markedly irregular as the result of a vegetative type of growth which prevents

TABLE 24 1

DIFFERENTIAL CHARACTERISTICS OF 11 STRAINS OF STREPTOCOCCI INVOLVED IN CENTRAL NERVOUS SYSTEM INFECTION *

	Hemolysis	Lactose	Sucrose	Salicin	Mannite	Raffinose	Inulin	Trehalose	Sdiol	Sodium Hippurate	Litmus Milk†
Carey Pig	Weakly beta	+	+	+	-	-	+	+	-	-	A sC,R
2169	Gamma	+	+	-	-	-	+	+	-	-	A sC,R
2180	Beta	+	+	-	-	-	-	+	-	+	A C,R
2191	Weakly beta	+	+	+	-	+	+	+	-	-	A C,R
Br 83	Gamma	-	-	-	-	-	-	-	-	-	No change
2225	Weakly beta	+	+	-	-	-	-	+	-	-	A C,R
6490	Gamma	+	-	+	-	+	+	+	-	-	A C
2299	Gamma	+	+	+	-	+	-	+	-	-	A sC
2298	Gamma	+	+	+	-	+	-	+	-	-	A sC
6B	Gamma	+	+	+	-	-	+	+	-	-	A
7B	Gamma	+	+	+	-	-	+	+	-	-	A

* From McNutt and Packer, 1913

† A = Acid
C = Coagulation
sC = Soft coagulation
,R = Partial reduction

The organisms were readily cultured from the brain, less frequently from the viscera, and occasionally from the joints They were Gram positive cocci occurring singly or in pairs and less frequently in chains Alpha hemolysis was produced on sheep blood agar and beta hemolysis on horse blood agar Growth in nutrient broth was poor The addition of 10 per cent horse serum improved growth Acid but no gas was produced in trehalose, salicin, inulin, lactose, dextrose, maltose, levulose, sucrose, and galactose Growth in sorbite, manite, raffinose, arabinose, dulcite, glycerin, in ositol, rhamnose, and xylose produced no change Some strains produced a partial reduction of 1 5000 methylene blue milk Acid was produced in litmus milk Starch was hydrolyzed The organism did not show a serological relationship to any of the Lancefield groups

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TABLE 24 1

Differential Characteristics of 11 Strains of Streptococci Involved in Central Nervous System Infection*

Carey Peg	Hemolysis	Lactose	Sucrose	Salicin	Mannite	Raffinose	Inulin	Trehalose	Sorbitol	Sodium Hippurate	Litmus Milk†
2169	Weakly beta	+	+	+	−	−	+	+	−	−	A sC$_2$R
2180	Gamma	+	+	−	−	−	+	+	−	−	A sC$_2$R
	beta	+	−	−	−	−	−	+	−	+	A C$_4$R
2191	Weakly beta	+	+	+	−	+	+	+	−	−	A C$_2$R
Br 83	Gamma	−	−	−	−	−	−	−	−	−	No change
2225	Weakly beta	+	+	+	−	−	−	+	−	−	A C$_4$R
6470	Gamma	+	−	+	−	+	+	+	−	−	A C
2299	Gamma	+	+	+	−	+	−	+	−	−	A sC
2298	Gamma	+	+	+	−	+	−	+	−	−	A sC
6B	Gamma	+	+	+	−	−	+	+	−	−	A
7B	Gamma	+	+	+	−	−	+	+	−	−	A

* From McNutt and Packer, 1943

† A = Acid
C = Coagulation
sC = Soft coagulation
R = Partial reduction

closing of the valves Emboli separate ly from these irregular masses and circulate in the blood stream, subsequently :king small arteries and frequently sing infarction of an area Infarctions he kidney are common If infarction of lore vital organ such as the wall of the rt or a section of the brain occurs, death ally results quickly

:ernkamp (1941) reported the occurrence of vegetative endocarditis in 19 cases urring over a 10 year period Of these, ptococci were isolated from 8, and *Erysipelothrix rhusiopathiae* from 11 cases e of the streptococcic cultures produced a hemolysis on horse blood agar plates o were nonhemolytic and one culture s not studied for this characteristic The ations of the lesions were not confined any particular valve Of the eight streptococcic cases the mitral valve alone was rolved in one case, the tricuspid alone in ree, the mitral and mural endocardium one, the tricuspid and mural endocardium in one, the tricuspid and pulonary in one, and the aortic alone in one Joint involvement in chronic streptococcic infections have been reported (Field al, 1954) Ray (1945) refers to the contion as 'navel ill when occurring in ung pigs This type of joint infection n be differentiated from that caused by *ysipelothrix rhusiopathiae* since the latr rarely occurs in pigs under 3 weeks of ;e Field et al described the joint lesions having turbid joint fluid with a high ll count but few organisms

Stomach ulcers were observed in 1,000 of 20,000 hogs at slaughter (Jensen and Frederick, 1939) The streptococci which were isolated from the ulcers were non hemolytic (alpha or *Str viridans*) on blood cultures *The organisms were believed to be primary invaders and able to cause stomach ulcers as a result of direct infection of the stomach tissues* Diagnosis would be difficult except at necropsy

TREATMENT

Generally, streptococci are quite susceptible to penicillin, although there is a danger of relapse if the treatment is not repeated within 4 to 6 days The sulfonamides also have some beneficial effects Any of the antibiotics which are effective against Gram positive bacteria can be used if given by injection into body tissues Oral administration is slower and less effective but may be of definite advantage in preventing a relapse

Heart and stomach lesions are difficult to diagnose and therefore difficult to treat Joint lesions respond very slowly to any treatment

PREVENTION

Good management with emphasis on sanitation is perhaps the only known preventative action which can be taken Antibiotics in the feed may prove to have some beneficial action Proper treatment of the navel with a disinfectant such as iodine at birth is an essential management practice (see Chapter 52)

Colibacillosis

Colibacillosis, white scours, diarrhea eonatorum, or diarrhea of baby pigs is an cute, sometimes highly fatal disease of ickling pigs characterized by a yellowish hite, watery diarrhea and often ac ompanied by a septicemia The disease, nder certain circumstances, is infectious nd spreads from pig to pig within a litter ut less frequently spreads from litter to tter within a farrowing house

ETIOLOGY

The causative agent or agents probably cannot be limited to one specific organism Enterobacteria such as *Escherichia coli*, members of the Klebsiella Aerobacter group, or *Salmonella choleraesuis* may be found to be the pathogenic agent or agents involved *The organism most commonly isolated from parenchymal organs in fatal septicemic cases is E coli*, a normal in

habitant of the digestive tract It is a small, Gram negative, oval to rod shaped bacillus which grows readily on common laboratory media and does not form spores It develops characteristic colonies with blackish centers on eosin methylene blue agar, is methyl red positive, indol positive, Voges Proskauer negative, and citrate nega tive For other biochemical reactions used to differentiate Enterobacteriaceae, see Ed wards and Ewing (1955) or Merchant and Packer (1956) *Aerobacter aerogenes* dif fers from *E coli* in size, shape, and growth characteristics Biochemically the main difference is that it is M R negative, indol negative, V P positive, and citrate positive *S choleraesuis* is discussed in Chapter 21

E coli exists in the digestive tract as part of the normal intestinal flora Certain strains are pathogenic for animals which have been chilled, particularly at the time of farrowing Pigs nursed by sows fed im properly balanced rations or too much feed and pigs maintained in wet, poorly cleaned farrowing pens with insufficient bedding are subject to infection with *E coli* Vitamin A and iron deficiencies also predispose young pigs to scours ' Man ninger (1939) is convinced that depletion of reserves of vitamins and minerals in the sow adversely affect intra uterine develop ment and the quality of the milk secretion This, in conjunction with poor manage ment, tends simply to increase numbers of facultative pathogens rather than to in crease their virulence Brucella infection in sows and extensive inbreeding were cited by McBryde (1931) as factors in pro ducing litters with lowered resistance

Infections with pathogenic strains of *E coli* probably are most severe if the infec tion takes place at the time of birth and before the piglet has received its first milk Infection also may be initiated by con tamination of the umbilicus at birth

CLINICAL SIGNS

Within the first few days after birth, pigs become listless and develop a watery, yellowish white diarrhea with a marked fetid odor Infected animals lose weight

rapidly and become very weak Such animals are not easily roused from the nest and usually do not live long The tails of infected pigs frequently become wet and coated with liquid or semi liquid feces If the pig lives this coating occasionally dries and forms a hard cake which may interfere with the circulation of the blood to such an extent as to cause a sloughing of a portion of the tail The death rate of infected untreated pigs varies but may be as high as 100 per cent

PATHOLOGICAL CHANGES

The principal lesion observed in pigs dying from colibacillosis is a mild to severe catarrhal enteritis characterized by a con gestion of both the anterior and posterior mesenteric vessels The stomach may con tain varying quantities of clotted milk but no lesions are noted The intestines may contain a watery, often gaseous yellowish white material Pneumonia pleuritis, and peritonitis may be present as compli cations Abscessed joints sometimes occur as a secondary infection after the disease appears to have abated

DIAGNOSIS

Diagnosis is based primarily on the presence of a moderately infectious yellow ish white diarrhea The absence of hemor rhagic lesions on the stomach and the limited degree of infectivity help to dis tinguish it from transmissible gastroenter itis of viral etiology (Chapter 6) Bacte rial culture of parenchymal organs, made at necropsy of scouring pigs in a moribund condition or shortly after death, will usually reveal a coliform bacterium if the disease is not of viral origin

IMMUNITY

The only highly susceptible period for *E coli* infection in the pig is the first few days of life Thereafter, whether or not the pig has been exposed to pathogenic strains of coliform organisms other than *S choleraesuis*, there is little occurrence of the disease This indicates the develop ment of a balance between the resistance

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Tuberculosis

In the Annual Report of the Bureau of Animal Industry for 1907, Mohler and Washburn stated "Indeed there is probably no disease of hogs, not even excepting hog cholera, which is causing heavier losses to the hog raiser than tuberculosis it must be considered as a general veterinary problem theoretically easy of solution which should receive the careful attention of all sanitarians" Tuberculosis is no longer such a serious problem in swine in the United States, but the decrease of the disease has not been as pronounced as for bovine tuberculosis The latter part of the statement by Mohler and Washburn, though made in the early 1900 s, may well be heeded today For the year ending June 30, 1957, there were slaughtered, under federal inspection, 20,142,195 cattle, of which only 3,310 carcasses (0 016 per cent) had lesions of tuberculosis In contrast, of 62,238,519 swine slaughtered during the same period, 1,841,623 carcasses (2 96 per cent) had lesions of tuberculosis (Pals, 1957) The percentage of infection is thus about 180 times as great for swine as for cattle, and this is cause for concern

There has been no direct campaign to eradicate tuberculosis in swine It was once

thought that the campaign to eradicate bovine tuberculosis, which was started in 1917, would result in a reduction of the prevalence of tuberculosis in swine in the United States However, the percentage of swine with tuberculous lesions continued to increase for a number of years, as shown in Table 25 1 The investigations of Van Es and Martin (1925) and those of Graham and Tunnicliff (1926) showed that most of the tuberculosis in swine was of avian origin

The decrease in prevalence of tuberculosis in swine in the United States is largely attributable to a lowering of the incidence of tuberculosis in poultry, which in turn is the result of the increasing practice of maintaining all pullet flocks of chickens The control of tuberculosis in swine is thus incidental to and a beneficial but secondary effect of a changing practice of poultry husbandry Perhaps a more rapid decline in tuberculosis among swine will occur if a direct and effective attack is made on tuberculosis in poultry

INCIDENCE

Because swine are not routinely tested with tuberculin, the only sources of information on the prevalence and geographic distribution of tuberculosis in this species are the data obtained from meat inspection

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[1] The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota

of the host and the invasion of facultative pathogens. Animals not previously exposed to *S. choleraesuis* remain susceptible to this organism for some time after the weaning period. When pigs develop to a weight of 60 lb. or more, the pathogens named above seldom are incriminated as primary causes of disease.

TREATMENT

Oral administration of drugs to baby pigs is generally considered somewhat impractical because of the number of treatments necessary and the difficulty in obtaining accurate dosage. The most convenient method of treatment of pigs is through medication of the sow with drugs which are subsequently secreted with the sow's milk. One gram of Terramycin injected intramuscularly into the sow has been found to be successful, whereas penicillin and streptomycin administered in a similar manner were not effective (Schipper et al., 1956). Good results have been obtained by injection of sulfamethazine into the baby pig at the rate of 1½cc of a 12½ per cent solution for each pound of body weight (Hokanson, 1958). One injection usually will suffice but may be repeated in 24 to 36 hours. Sulfathalidine (phthalyl sulfathiazole) administered in 0.25 Gm. doses to pigs between the ages of 3 to 10 days, and 0.5 Gm. doses every second day for those over 10 days resulted in 95 per cent recovery as compared to 85 per cent mortality in untreated controls (Edmonds, 1916). Treatment of associated anemia with injectable iron in the form of iron dextran compound is very helpful. Treatment of the umbilicus with iodine at the time of birth is effective in controlling joint infection.

EPIZOOTIOLOGY AND CONTROL

The organism is readily spread from pig to pig by the consumption of infected feces. As the disease spreads, the organism develops greater pathogenicity or increased numbers with a resulting increase in pig morbidity and mortality. Control is best effected through management. According to McBryde (1934), if pigs were removed from the sow immediately after farrowing, kept away for 4 hours, and then returned to the sow to receive colostrum the disease did not occur. Ample dry bedding, elimination of drafts, provision of heat lamps in chilly weather, clean dry pens, and a good sow ration (see Chapters 36 and 51) are of major importance.

REFERENCES

BRYANT, J. B. 1945 Streptococcic septicemia in swine. Jour. Amer. Vet. Med. Assn. 106:18.
EDMONDS, E. V. 1916 Further observations on the use of sulfathalidine phthalylsulfathiazole in the treatment of enteritis in suckling pigs. No. Amer. Vet. 27:561.
EDWARDS, P. R. AND EWING, W. H. 1955 Identification of Enterobacteriaceae. Burgess Publishing Co. Minneapolis, Minn.
FIELD, H. I., BUNTAIN, D. AND DONE, J. T. 1954 Studies on piglet mortality. 1. Streptococcal meningitis and arthritis. Vet. Rec. 66:453.
HOKANSON, J. F. 1958 Personal communication.
JENSEN, L. B. AND FREDERICA, L. D. 1939 Spontaneous ulcer of the stomach in several domestic animals. Jour. Amer. Vet. Med. Assn. 95:167.
KERNKAMP, H. C. H. 1911 Endocarditis in swine due to Erysipelothrix rhusiopathiae and to streptococci. Jour. Amer. Vet. Med. Assn. 108:152.
McBRYDE, C. N. 1934 Acute enteritis in young pigs due to infection with colon group. Jour. Amer. Vet. Med. Assn. 84:36.
McNUTT, S. H. AND PACKER, R. A. 1913 A study of some cases of streptococcic infection in swine. Vet. Student 6:68.
MANNINGER, R. 1939 Diseases of the digestive system of young pigs. Vet. Rec. 51:1159.
MERCHANT, I. A. AND PACKER, R. A. 1956 Veterinary Bacteriology and Virology. Iowa State College Press, Ames, Iowa.
RAY, J. D. 1945 Enteritis and mixed infections of swine. Vet. Med. 40:48.
SCHIPPER, I. A., BUCHANAN, M. L. AND EVELETH, D. F. 1956 Swine enteritis. I. Terramycin in the treatment of diarrhea of suckling pigs. Jour. Amer. Vet. Med. Assn. 128:92.

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CHAPTER 25

Tuberculosis

In the Annual Report of the Bureau of Animal Industry for 1907, Mohler and Washburn stated "Indeed there is probably no disease of hogs, not even excepting hog cholera which is causing heavier losses to the hog raiser than tuberculosis it must be considered as a general veterinary problem theoretically easy of solution which should receive the careful attention of all sanitarians" Tuberculosis is no longer such a serious problem in swine in the United States, but the decrease of the disease has not been as pronounced as for bovine tuberculosis The latter part of the statement by Mohler and Washburn, though made in the early 1900 s, may well be heeded today For the year ending June 30, 1957, there were slaughtered, under federal inspection, 20,112,195 cattle, of which only 3,310 carcasses (0 016 per cent) had lesions of tuberculosis In contrast, of 62 238 519 swine slaughtered during the same period, 1,811,623 carcasses (2 96 per cent) had lesions of tuberculosis (Pals, 1957) The percentage of infection is thus about 180 times as great for swine as for cattle, and this is cause for concern

There has been no direct campaign to eradicate tuberculosis in swine It was once

thought that the campaign to eradicate bovine tuberculosis, which was started in 1917, would result in a reduction of the prevalence of tuberculosis in swine in the United States However the percentage of swine with tuberculous lesions continued to increase for a number of years as shown in Table 25 1 The investigations of Van Es and Martin (1925) and those of Graham and Tunnicliff (1926) showed that most of the tuberculosis in swine was of avian origin

The decrease in prevalence of tuberculosis in swine in the United States is largely attributable to a lowering of the incidence of tuberculosis in poultry which in turn is the result of the increasing practice of maintaining all pullet flocks of chickens The control of tuberculosis in swine is thus incidental to and a beneficial but secondary effect of a changing practice of poultry husbandry Perhaps a more rapid decline in tuberculosis among swine will occur if a direct and effective attack is made on tuberculosis in poultry

INCIDENCE

Because swine are not routinely tested with tuberculin the only sources of information on the prevalence and geographic distribution of tuberculosis in this species are the data obtained from meat inspection

TABLE 25 1

Incidence of Tuberculosis in Swine in the United States as Determined by Necropsy Inspection in Abattoirs Under Federal Supervision*

Year	Number Slaughtered	Per Cent Tuberculous†	Per Cent Condemned‡
1907	31,815,900	1 34	0 20
1912	34,966,378	4 69	0 12
1917	40,210,847	9 89	0 19
1922	34,416,439	16 38	0 20
1927	42,650,443	13 54	0 14
1932	45 852,422	11 38	0 08
1937	36,226,309	9 48	0 08
1942	50,133,871	7 96	0 026
1947	47,073,370	8 50	0 023
1952	63,823,263	4 40	0 015
1956	66,781,940	4 76	0 010

* Data compiled from Year Book, USDA (1922), Feldman (1955), Pickard (1952), and from USDA (1956)

† Includes all carcasses with evidence of tuberculosis, varying in extent from only small foci in cervical lymph nodes to generalized involvement

‡ Includes only carcasses with evidence of generalized tuberculosis

records Reference to Table 25 1 shows that on this basis there was in the United States an increase in the rate of infection until 1922, during which year 16 38 per cent of all swine slaughtered under federal supervision had tuberculous lesions, in 0 20 per cent the disease was so extensive that the entire carcass was condemned Since 1922 there has been a gradual decline, by 1956 the incidence had lowered to 4 76 per cent with only 0 01 per cent having evidence of generalized tuberculous disease

Geographically, there is in the United States a wide variation in the prevalence of tuberculosis in swine The infection appears to occur most frequently in the north central states Feldman (1936) recorded that the retention of swine for tuberculosis in South St. Paul and Chicago was 15 09 per cent and 11 35 per cent, respectively, as compared to 6 8 per cent and 1 9 per cent for St. Louis and Kansas City In 1951, in federally supervised abattoirs in Cleveland, Detroit, and South St. Paul, the percentage of swine carcasses with tuberculous lesions was 8 17, 8 01, and 6 6 per cent, respectively, in contrast to 1 57 and 1 38 per cent for Fort Worth and Kansas

City, respectively (Feldman, 1952) Pickard emphasized the greater incidence of tuberculosis in swine in the north central states than in other sections of the United States by showing that for the year ending June 30, 1952, one of 12 swine slaughtered in abattoirs in Michigan had lesions of tuberculosis, as compared to one of 190 slaughtered in Georgia During 1955, in the United States only 3 2 per cent of 61,370,474 swine slaughtered under federal supervision, had evidence of tuberculosis In that year the incidence ranged from 3 5 per cent for the area including Iowa, Minnesota, Nebraska, North Dakota, and South Dakota to 1 3 per cent for the area including Florida, Georgia, South Carolina, North Carolina, and Virginia (Pals, 1957)

Since most of the tuberculosis in swine in the United States is of avian origin, it is reasonable to expect that the prevalence of the disease among swine is greater in the north central states where tuberculosis among chickens is greater (Pickard, 1952). However, in evaluating the geographic prevalence of tuberculosis based on meat inspection records, it should be remembered that swine may be transported to abattoirs at great distances from their origin For example, Ranney (1955) reported that, in 1954, shipments of swine with a high incidence of tuberculosis in Philadelphia could be traced to one of the central states, and in 1955 tuberculous swine reported in Albany, New York, were eventually found to have originated in the middle west.

Reports from various parts of the world indicate that tuberculosis in swine is recognized as a problem and that its prevalence perhaps reflects inversely the success of controlling tuberculosis in other species. In Great Britain (Thornton, 1919) it was said that tuberculosis in swine was due largely to the feeding of dairy by products In one abattoir, 11 0 per cent of 1,500 000 swine from all parts of Great Britain had tuberculous lesions, the majority of which were localized in the submaxillary lymph nodes. In France (Comminy, 1953), at the

abattoir in Le Havre, the incidence in 1946 was less than 0 1 per cent, but this had in creased to 10 per cent in 1952 as the result of an increase in the practice of feeding dairy by products In Switzerland (Lanz, 1955) there had been a decrease from 2 32 per cent in 1946 to 1 32 per cent in 1953, due to better control of tuberculosis in cat tle In Germany (Hubner, 1956) the meat inspection statistics showed a slight de crease in the percentage of swine with tuberculous lesions, from 2 9 per cent in 1950 to 2 2 per cent in 1954, which is at tributed to efforts to reduce the incidence of tuberculosis in cattle

In Denmark (Bendixen, 1950, 1956), ac cording to official figures, the extent of tuberculosis among swine fell gradually from about 4 5 per cent in 1925 to 1 or 2 per cent in 1944, coincidently with the decrease of bovine tuberculosis There has continued to be a small percentage of swine (0 44 per cent in 1955) with local ized lesions due to avian tubercle bacilli

In Egypt (Elian et al, 1953) at the abattoir in Cairo, 62 (2 per cent) of 3,143 swine had lesions of tuberculosis, of which 34 were due to human type tubercle ba cilli Tuberculosis is said to be common in swine in South Africa and is due pri marily to bovine sources (Fourie et al, 1950, Robinson, 1955) In Australia, (Al biston et al, 1954) surveys made in Vic toria indicate a drop in the incidence of tuberculosis in swine in a 40 year period ending in 1951 from about 4 0 or 5 0 per cent to 1 5 or 2 5 per cent A slight in crease in recent years was attributed to feeding of more dairy by products In Queensland the incidence of tuberculosis in swine dropped from 7 per cent in 1910 to 1 per cent in 1953, owing to control of the disease in cattle. In South Australia, however, the rate increased from 1 or 2 per cent in 1936 to 7 per cent in 1954, owing to expansion of the poultry industry

Data on the prevalence of tuberculosis in swine as compiled from meat inspection records may be misleading because the diagnoses are made on the basis of macro scopic appearance of lesions. A certain number of tuberculous infections will es cape detection because the lesions are not grossly visible Avian tubercle bacilli have been isolated from tonsils (Feldman and Karlson, 1940) and from lymph nodes (McCarter et al, 1935) of apparently nor mal swine as well as from grossly normal lymph nodes of carcasses that were 'passed for food' after removal of localized lesions (Feldman, 1936) Furthermore, in studies in the United States and Canada where presumably tuberculous lymph nodes of swine were collected at abattoirs and ex amined bacteriologically, a varying but high percentage failed to yield tubercle bacilli, as shown in Table 25 2 Similar observations have been made by workers in Australia (Clapp, 1956 Pullar and Rush ford, 1954), England (Cornell and Grif fith 1930 Cotchin, 1910?) Denmark (Plum, 1916) and Germany (Baumann et al, 1955, 1956) to mention a few The failure to demonstrate tubercle bacilli in lesions which appear grossly to be tuber culous may be due to (1) inadequacy of present day methods for isolating tubercle bacilli (2) occurrence of healed processes that contain no viable tubercle bacilli or (3) causation of the lesions by some micro organism other than tubercle bacilli such as Corynebacterium equi, discussed later in this chapter

SOURCES OF INFECTION AND CONTROL

Swine are susceptible to infection with Mycobacterium tuberculosis var bovis, My cobacterium tuberculosis var hominis, and Mycobacterium avium The occurrence of tuberculosis in swine is therefore related to the opportunity for direct or indirect contact with tuberculous cattle, human beings and fowl and to the prevalence of tuberculosis in these species

The bovine tubercle bacillus is not a common cause of tuberculosis in swine in localities where the disease in cattle is con trolled by a campaign of eradication In the United States and Canada, for example, bovine tubercle bacilli are rarely found in lesions of swine, as shown in Table 25 2. Elimination of bovine tuberculosis in the

primary host thus serves to control the disease in swine. However, the occasional finding of bovine tubercle bacilli in swine is a reminder that the disease in cattle is a constant threat. Efforts to eradicate bovine tuberculosis should not be diminished.

Where tuberculosis does occur in cattle, *the infection may be transmitted to swine* by feeding of unpasteurized milk and dairy by-products. This danger was recognized in Denmark where, in 1898, compulsory pasteurization was introduced, not primarily to protect the human population but to prevent transmission of bovine tubercle bacilli to calves and pigs in by-products of dairies (Bang, 1899). It has been shown that feces of tuberculous cattle may contain *an obvious hazard where swine and cattle are maintained in a common feed lot* (Schroeder and Mohler, 1906). Tuberculous metritis in cattle and consequent abortion create opportunities for infecting swine. It is obvious that swine should not be exposed to infected cattle. The practice of feeding swine the offal

from abattoirs or feeding uncooked garbage is obviously unwise, since such material may contain tuberculous material from beef carcasses.

The human type of tubercle bacillus is occasionally isolated from tuberculous lesions in swine. No person known to have active tuberculosis should be permitted to have any contact with swine or other animals. In the Philippines, *Topacio* (1933) found that each of 11 cultures isolated from swine was of human origin. This was attributed to the custom of permitting swine to run at large in rural areas where the incidence of tuberculosis among people was high and where standards of sanitation were low. In Finland, *Svandberg* (1935) examined tuberculous lesions *from 60 swine from abattoirs during 1931 and 1932 and found that 21 had human tubercle bacilli and 10 had bovine type. No avian strains were demonstrable.* (Specimens from 11 animals failed to yield any tubercle bacilli, and in 12 instances the examination was incomplete) These results were said to reflect the high tuber

TABLE 25 2

SUMMARY OF DATA COMPILED FROM REPORTS IN NORTH AMERICA ON THE OCCURRENCE OF TUBERCLE BACILLI IN TUBERCULOUS LYMPH NODES OF SWINE

(Specimens obtained from abattoirs under federal supervision)

Author	Date*	Origin of Swine	Number of Specimens	Type of Tubercle Bacillus, Per Cent			
				Avian Only	Mammalian Only	Mixed	None†
Van Es and Martin	1925	Nebraska	248	74 6	4 4	5 6	15 4
Van Es and Martin	1925	Michigan	14	92 9	none	7 1	none
Graham and Tunnicliff	1926	Illinois	85	60 0	4 8	8 2	27 0
McCarter and associates	1935	Wisconsin	61	65 5	none	none	34 5
Feldman	1936b	Southeastern Minnesota	30‡	80 0	6 6 (bovine)	none	13 3
Crawford	1938	North Central States	36‡	58 3	41 6 (bovine)	none	none
Feldman	1939	Minnesota	75§	46 6	16 0 (human)	none	37 3
Feldman and associates	1940	Minnesota	89	61 8	none	none	38 2
Mitchell and associates	1934	Canada	96	39 5	none	none	61 5
Pullin	1946	Eastern Canada	232	44 8	0 9 (bovine)	none	54 3
Banker	1946	Alberta	102	88 0	1 0 (bovine)	none	11 4

* In several papers it is indicated that the work was done from 1 to 2 years prior to publication.
† Tubercle bacilli not demonstrated by cultural or by animal inoculation tests.
‡ Selected cases of generalized tuberculosis, none of the specimens were portions of lung, liver, or spleen.
§ Garbage-fed swine.

culosis morbidity in the human population and the low incidence of tuberculosis in poultry in Finland at that time

Uncooked garbage is a potential means of transmitting tuberculosis to swine But ler and Marsh (1927) found tuberculous lesions in cervical and mesenteric lymph nodes in 26 of 80 swine fed uncooked gar bage from a hospital with a number of tuberculous patients Material from some of these affected animals was studied and found to contain human type tubercle bacilli Feldman (1939) recorded that of 264 garbage fed swine, 75 (28 4 per cent) were found at the time of slaughter to have tuberculous lesions Of these, 47 were found to contain tubercle bacilli of which 35 were of avian and 12 were of human type It was concluded that garbage may *contain the offal of tuberculous chickens* and also that material from tuberculous patients is not properly disposed of Per haps one way to encourage the isolation of tuberculous patients and to insist on hy gienic disposal of wastes is to show the *economic loss that may result from infect ing food producing domestic animals*

The frequent occurrence of avian tuber cle bacilli in lesions limited to the cervi cal and mesenteric lymph nodes in natur ally infected swine indicates that infection usually occurs by ingestion Graham and Tunnicliff (1926), who were concerned with apparent irregularities in the control of bovine tuberculosis in Illinois, investigated the possibility that fowl may be a source of tuberculous infection to other animals. The results of their experiments on trans mission of avian tuberculosis to swine may be summarized as follows (1) Lesions in swine are usually local and confined to lymph nodes of the digestive tract, particu larly the mesenteric nodes, (2) swine may be easily infected by feeding of grain con taminated with feces of tuberculous chick ens as well as by feeding of organs of tuberculous fowl, (3) swine may be in fected by occupying an area in which tuberculous chickens have previously been confined (4) the infection may be trans mitted from swine to swine

Schalk and co workers (1935) obtained similar results in their extensive studies in North Dakota Of particular importance was their observation that swine contracted tuberculosis when placed on ground that had not been occupied by tuberculous chickens for the previous 2 years Viable and pathogenic avian tubercle bacilli were found in the soil and litter of a chicken cage after 4 years Schalk and co workers concluded that soil contaminated by feces of tuberculous fowl is the most important source of infection for swine No success was obtained in controlling the disease merely by use of the tuberculin test and elimination of reactors, because the soil remained infective They recommended that an ideal program to control avian tuberculosis is to rear young birds on clean ground and to dispose regularly of all fowl more than 1 year old Lee (1956) also emphasized the importance of elimi nating older chickens in efforts to control tuberculosis in swine He found that in Iowa, *during the period from 1946 to 1949 only 0 24 per cent of 33 769 chickens in all pullet flocks were tuberculin positive* as compared to 11 52 per cent for 5 382 hens 2 years old or more

Wild birds may be incriminated as a *source of avian tuberculosis in swine* Gra ham and Tunnicliff (1926) showed that the disease could be produced in pigs by the feeding of naturally infected sparrows Schalk and co workers also considered wild birds to be a *possible means of spreading* avian tuberculosis In a herd of pigs having no contact with poultry, McDiarmid (1956) found that some animals reacted to avian tuberculin and were found to have tuberculous lesions The source of infection was attributed to tuberculous sparrows found in the vicinity

The close contact of swine in yards and feeding pens provides opportunity for transmission of tuberculosis from animal to animal The occurrence of intestinal lesions, as shown in Fig 25 1, allows spread of tubercle bacilli in feces Graham and Tunnicliff (1926) found that rectal scrap ings of some tuberculous swine contained

viable avian tubercle bacilli. Feldman and Karlson (1940) and Pullar and Rushford (1954) have demonstrated avian tubercle bacilli in the tonsils of pigs. The latter workers suggested that this may be a source of infection to other animals. Pulmonary, uterine, and mammary tuberculous lesions in swine constitute sources of infection to other animals. Plum and Slyngborg (1938) examined the lungs of 96 swine with pulmonary tuberculosis and isolated bovine tubercle bacilli from the bronchial mucus of 23. In the same report Plum and Slyngborg mentioned that about 28 per cent of 1,700 sows had tuberculous lesions, and that 15 per cent had involvement of the uterus, two to three times as many had tuberculous mastitis. In all the cases the infection was the bovine type

IMMUNIZATION OF SWINE WITH BACILLE CALMETTE GUERIN (BCG)

The control of tuberculosis in swine by immunization with BCG has been advocated, especially where infection due to *bovine tubercle bacilli* is serious. A brief account of using BCG in swine in Chile was given by Sanz (1930), who reported that over a 3-year period, 993 pigs were vaccinated at birth and separated from their dams. In none of the vaccinated swine did tuberculosis develop, although they were on farms where bovine tuberculosis was present. The only swine that died of tuberculosis were the older, unvaccinated animals. In 1948 Girard reported that in Madagascar, where bovine tuberculosis is common, immunization of swine with BCG is a useful procedure. In the period from 1930 to 1942, 1,800 pigs were vaccinated at birth, with good results. The vaccination permitted the successful raising of swine where previously the occurrence of bovine tuberculosis had caused serious losses in herds of swine.

However, the results of experimental studies with suitable controls have shown that, in swine, BCG affords little if any appreciable protection against *tuberculosis* due to bovine tubercle bacilli. Jundell and Magnusson (1931) in Sweden vaccinated 16 pigs, 11 to 14 days old; eight were given 10 mg. of BCG intramuscularly and eight were given 10 mg. orally on 3 successive days. Two months later the vaccinated animals plus six control animals were fed tuberculous bovine udder tissue. In 19 to 22 weeks, all of the animals were slaughtered and all were found to have extensive tuberculous disease. It was concluded that there was no evidence of protection due to the use of BCG.

In this country, Hayes *et al.* (1932) conducted a number of experiments in which BCG was administered to swine by various routes. Comparison of the extent of *tuber-*

FIG 25.1—Submucosal tuberculous lesion due to avian tubercle bacilli in the intestinal tract of a pig. The lesion appears to be extending toward the surface, where it may ulcerate and discharge bacilli into the lumen. The diffuse cellular proliferation with little necrosis is typical of avian tubercle bacillus infection in swine. Hematoxylin-eosin X 50

culous disease in vaccinated animals and in the controls led to the conclusion that one injection of BCG of 100 mg given by the subcutaneous, the intramuscular, the intradermal, or the intravenous route failed to protect against generalized tuberculosis induced by intravenous infection or by feeding infective material of bovine origin Also, three successive oral doses of 100 mg of BCG failed to provide any resistance to infection These workers also showed that the presence of an inoculation lesion at the injection site of BCG failed to have any premunitive effect This report of carefully controlled experiments appears to be a conclusive demonstration of the ineffectiveness of BCG for the control of tuberculosis in swine

TUBERCULOSIS LIKE LESIONS ASSOCIATED WITH CORYNEBACTERIUM EQUI IN LYMPH NODES OF SWINE[2]

As mentioned previously, a relatively high percentage of localized tuberculous lesions in lymph nodes of swine have failed to yield tubercle bacilli when examined by bacteriologic or animal inoculation procedures Referring to Table 25 2 it is seen that this is a common experience The failure to demonstrate tubercle bacilli may be due to inadequate technic, to healing of the lesion, or to the fact that the lesion was not the result of infection by tubercle bacilli With respect to the latter possibility, special mention must be made of the occurrence of *Corynebacterium equi* in localized lesions that cannot be easily differentiated from tuberculous processes either macroscopically or histologically

Holth and Amundsen (1936) in Norway reported that of 162 tuberculous lymph nodes from swine only 103 yielded tubercle bacilli (97 were typed, of which there were 80 avian, 16 human, and one bovine strain) Of the other 59, there were 38 that contained a variably acid fast coccobacillus The acid fastness, however, was not constant and was lost on subculture The presence of this microorganism in

localized tuberculosis like lesions in swine was soon confirmed by other Scandinavian workers Bendixen and Jepsen (1938) in Denmark showed that the microorganism in question was actually *C equi*, which was known to be the cause of a purulent pneumonia in horses The lesions in swine are called Holth s processes and the microorganism is referred to as Holth s bacillus or *Corynebacterium Magnusson Holth'* in some reports

The microorganism is a diphtheroid which grows well on ordinary culture medium forming smooth colonies with a characteristic pink color *C equi* has been found in the soil, as well as in pathologic conditions in various species of domestic animals The microorganism is not pathogenic for the usual laboratory animals

In Wisconsin, McCarter et al (1935) recorded as an incidental finding that cultures of an orange pink diphtheroid' were isolated from 21 of 61 tuberculous cervical lymph nodes of hogs but were not found in lymph nodes of nontuberculous swine In Minnesota, Karlson et al (1940) described the isolation of *C equi* from tuberculous lymph nodes as well as from normal submaxillary lymph nodes of swine In the diseased nodes the microorganism was occasionally found alone, but in most instances it was associated with avian tubercle bacilli These workers attempted without success to reproduce lymphadenitis in swine with cultures of *C equi* It was concluded that the etiologic significance of *C equi* in tuberculosis like lesions in swine was doubtful In England, Cotchin (1940b) also found *C equi* in tuberculous cervical lymph nodes of pigs as well as in normal animals He too doubted that this microorganism had any pathogenic significance in swine However, various investigators in the 1910 s and 50 s have considered that *C equi* is of importance because it is often found either alone or with tubercle bacilli in localized lesions of the head and neck in swine

Plum (1946) in Denmark studied a large number of tuberculous lymph nodes from swine and concluded that it is difficult for inspectors in abattoirs to differen

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[2] A more complete discussion of this problem and additional references may be found in the monograph by Ottosen (1945)

tiate so-called Holth's processes from lesions caused by tubercle bacilli. This problem has been recently recognized in South Africa (Robinson, 1955) and in Australia (Tammemagi, 1953; Clapp, 1956) as well as in various European countries. Ottosen (1915) has shown that *C. equi* occurs more frequently in the soil of hog pens than elsewhere. As a preventive measure Ottosen recommended that the use of certain pens for pigs should be avoided if cultures of the soil reveal *C. equi*.

TUBERCULIN TEST

Of historical interest are the studies of Schroeder or Mohler (1906) on the subcutaneous or thermic tuberculin test in swine. Animals were confined in crates to prevent exercise, which caused variations in temperature. Rectal temperatures were recorded every 2 hours on the day preceding and every 2 hours on the day following a subcutaneous injection of 0.5 ml. of tuberculin. A positive reaction was recorded when there was an increase in temperature of 1° F. Only two failures were reported in 58 animals. One reactor had no visible lesions, and one tuberculous pig failed to react.

The intradermal test, usually on the ear, is now employed. Since swine are susceptible to infection with avian and with mammalian tubercle bacilli, it is advisable to use avian and mammalian tuberculin. Van Es (1925) warned that, if only mammalian tuberculin is used for testing swine, a considerable number of cases will escape detection and that for dependable results avian tuberculin must also be used. Van Es (1925) and Luke (1953) have suggested that for swine, avian and mammalian tuberculin may be mixed and given in a single injection. However, a positive reaction to such a test would not indicate whether avian or mammalian infection were present.[3]

Feldman (1938a) has recommended the use of 0.2 ml. of 25 per cent Old Tuberculin applied into the dermis on the dorsal surface of the ear slightly anterior to the base. A positive reaction is indicated in 24 hours by a flat reddish swelling up to 3 cm. in diameter, which in 48 hours reaches its maximal intensity. At this time the erythema and swelling are more pronounced; the central area becomes hemorrhagic and ulceration may occur. McDiarmid (1956) described a means of testing swine in which restraint is not necessary. While the animals are feeding from a trough, 0.1 ml. of tuberculin is injected at a right angle into the skin at the junction of ear and neck, a needle only 3.5 mm. long being used. With this short needle most of the tuberculin is said to be deposited in the skin. By use of a syringe in each hand it is possible to inject avian tuberculin on one side and mammalian on the other. Reactions are recorded in 48 hours. A positive reaction varies from "puffy" edema to inflammation, with purple discoloration and necrosis. McDiarmid used Weybridge PPD, which, according to Paterson (1949), has 3.0 mg. of protein per ml. for mammalian and 0.8 mg. of protein per ml. for avian tuberculin.

Lanz recommended injecting the tuberculin in the skin of the back about 10 to 20 cm. posterior to the shoulders and slightly to the right of the midline. This was said to be easier and less time-consuming than trying to use the ear. A dose of 0.1 ml. of Purified Protein Derivative, PPD, (as used for cattle in Switzerland) is injected intradermally. A positive reaction reaches its peak in 72 hours and consists of a painful erythematous swelling 22 to 35 mm. in diameter. No false or atypical reactions were found among 316 animals, as determined by necropsy examination.

Luke (1952) described observations on the tuberculin test in 100 sows, 3 or more years old, using avian and mammalian tuberculin intradermally in the ear and recording the results in 24 hours. A positive reaction was ascribed when there was an increase of 2 mm. in thickness of the

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[3] For an account of early studies on the specificity of avian and mammalian tuberculin in swine, the report by Bang (1917) may be consulted.

skin at the site of injection Of 39 reactors, only 22 had visible lesions, chiefly of lymph nodes of the digestive tract Tubercle bacilli were demonstrable in only three of the 22 animals with lesions, and each was a mammalian strain Lesions considered to be tuberculous were found in eight nonreactors, but tubercle bacilli were apparently not demonstrable in these In Luke's opinion, there is a large percentage of error in the tuberculin test in swine due to nonspecific sensitivity or to residual sensitivity from healed tuberculous lesions Negative reactions in animals with lesions may, according to Luke, be ascribed to the "ability of the pig to overcome and apparently sterilise existing lesions [4]

The reliability of the tuberculin test in swine was examined by Pullar and Rushford (1954) in Australia These workers tested 531 animals with avian and with mammalian tuberculin given intradermally in 0 1-ml amounts at the base of the ear Reactions were recorded in 72 hours An increase in thickness of skin of 100 per cent or more (more than 4 mm) was considered to be a positive reaction All animals were subjected to special examination in the abattoir Tuberculous lesions from nonreactors as well as from reactors were collected for bacteriologic study Only 36 (6 8 per cent) of the 531 animals reacted positively to tuberculin, and only two of the 36 reacted to mammalian tuberculin Tuberculous lesions were found in eight nonreactors, five of which were found to have tubercle bacilli According to the authors, the presence of such false negative tuberculin reactions in swine indicates the need of repeated tests in a herd A third of the reactors had no macroscopic evidence of tuberculosis Of particular importance was the finding that tubercle bacilli were demonstrable in only half of the lesions designated as tuberculous by gross inspection

PATHOLOGIC ANATOMY[5]

As seen in the abattoirs, tuberculous lesions in swine are usually limited to lymph nodes of the pharyngeal and cervical regions and of the mesentery The lesions vary in appearance from small yellowish white caseous foci a few milli meters in diameter to diffuse enlargement of the entire node The disease may be localized in one group of nodes or may involve a number of lymph nodes along the digestive tract

Gross differentiation between tuberculous adenitis due to avian and that due to mammalian tubercle bacilli is difficult but in general there are some features characteristic of each In an infection of avian origin, the lymph nodes may be enlarged and firm with no discrete purulent foci or there may be one or more soft caseous areas with indistinct borders Calcification is rarely demonstrable The cut surface of the lesion has a neoplastic appearance with a few caseous foci Although there may be diffuse fibrosis, there is little tendency to encapsulation Relatively large areas of caseation may be present and occasionally will involve the entire lymph node The lesions due to tubercle bacilli of the avian type are generally not easily enucleated In contrast, when the infection is of mammalian origin (either bovine or human), the lesions tend to be well encapsulated and are relatively easy to separate from the surrounding tissue In addition, calcification is prominent in lesions due to infection with mammalian tubercle bacilli The individual foci appear to be discrete and caseous These distinctions are by no means absolute, and there are many variations in the gross appearance of tuberculous lesions in lymph nodes of swine

Clapp (1956) examined, by bacteriologic procedures, 420 lymph nodes (mostly submaxillary) designated as tuberculous on

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[4] Luke (1955) has studied various aspects of the tuberculin test in swine including the specific effect of tuberculin on the white blood cell count The reader is referred to Luke's paper for an account of these studies

[5] Detailed discussions of the pathologic anatomy of tuberculosis in swine may be found in the paper by Pallaske (1931) and in the monograph by Feldman (1938a)

meat inspection. There was some correlation between the gross appearance and the etiology. Localized lesions that were not easily enucleated and large, dry, calcareous processes involving an entire lymph node were usually due to avian tubercle bacilli. Indistinctly mottled and streaked lesions, large encapsulated purulent abscesses, and lesions that could be easily enucleated were usually not due to tubercle bacilli. Some of these yielded *C. equi*, which Clapp considered as important in producing tuberculosis-like lymphadenitis in swine. In the series of 420 specimens, only five were from cases of generalized tuberculosis in swine and each of the five was due to bovine tubercle bacilli.

Microscopically, the changes induced in swine tissues by avian tubercle bacilli are ˙ accerued by diffuse proliferation of epithelioid cells and giant cells. There may be some necrosis and calcification, especially in older lesions, but these changes are not usually prominent. Proliferation of connective-tissue elements accompanies the process, but there is little or no tendency to form a well-defined wall by fibrosis. In contrast, lesions due to mammalian tubercle bacilli have a pronounced tendency to become encapsulated by a well-developed zone of connective tissue. In addition, there is early caseation and marked calcification. These differences are illustrated in Figure 25.2.

Generalized tuberculosis in swine is not commonly seen. In most instances it is due to infection with bovine tubercle bacilli, but it may be due also to the avian type (Feldman, 1938b; Crawford, 1938). The extent and character of generalized involvement vary from the occurrence of a few small foci in several organs to extensive nodular processes involving the liver, spleen, lungs, kidneys, and many lymph nodes. Generalized lesions due to infection with avian tubercle bacilli tend to be diffuse. The cut surface is usually smooth, and there is no great tendency toward encapsulation by fibrosis. There may be foci of caseation, but calcification is not pronounced. Lesions resulting from infection

with mammalian tubercle bacilli, however, are likely to be discrete, caseous, and well circumscribed by fibrosis. Calcification is prominent. Figure 25.3a and b shows portions of liver and lung from a hog with generalized tuberculous disease from which bovine tubercle bacilli were isolated.

DIAGNOSIS

A clinical diagnosis of tuberculosis in swine is presumptive at best. In the majority of cases the tuberculous lesions are limited to small foci in a few lymph nodes of the digestive tract. It is difficult to conceive that such nonprogressive morbid changes may elicit signs detectable by physical examination. In cases of extensive tuberculous infection there may be signs that are suggestive of an infectious disease, but the symptoms and changes are not sufficiently characteristic to establish a diagnosis of tuberculosis.

When there is rapidly disseminating tuberculous disease, there may be indications of generalized infection such as elevated temperature, anorexia, and loss of weight. As the disease progresses, symptoms such as dyspnea, diarrhea, and meningismus may develop, depending on the extent of involvement in certain organs. Tuberculous enlargement of lymph nodes may interfere with functions of adjacent organs. Dysphagia, dyspnea, and coughing result from greatly enlarged lymph nodes in the pharynx, in the neck, in the mediastinum, or in the hilus of a lung. Similarly, signs of digestive disturbances may be the result of greatly enlarged tuberculous lymph nodes of the alimentary tract. Pressure on motor nerves by enlarged lymph nodes will result in paralysis. Visible or palpable tuberculous lesions in swine include enlargement of peripheral lymph nodes, arthritis, orchitis, and mastitis. Tuberculous processes in these situations may ulcerate and form draining sinuses. Tuberculous metritis may give rise to vaginal discharge.

The necropsy and histopathologic appearance of tuberculosis in swine has been described. Although these morbid changes

are sufficiently characteristic to permit a tentative diagnosis of tuberculosis, they are not specific. The great similarity between localized tuberculous lesions and those associated with *C equi* has already been discussed Also, it may be difficult to grossly differentiate chronic granulomatous lesions due to various infectious agents parasitic nodules, and neoplasms

The tuberculin test for the diagnosis of tuberculosis in swine appears to be a useful procedure on a herd basis Of the various technics described for this test in swine the operator should select the method which, by experience proves to be most suitable Separate tests with avian and mammalian types of tuberculin must be made A number of investigators have found that some tuberculous swine may

fail to react to the intradermal tuberculin test It is advisable, therefore that repeated tests be made in a herd where reactors have been found and have been eliminated

The mere demonstration of acid fast bacilli in exudates or in lesions may be misleading Some workers have recorded that *C equi* is acid fast in smears of necrotic material from lymph nodes of swine (Ottosen, 1945) Acid fast microorganisms other than tubercle bacilli have been isolated from swine (Baumann *et al*, 1956 Karlson and Feldman, 1940)

The characteristic pathologic feature of tuberculosis in swine and the presence of acid fast microorganisms in such lesions provide important indications on which to base a diagnosis of tuberculosis However
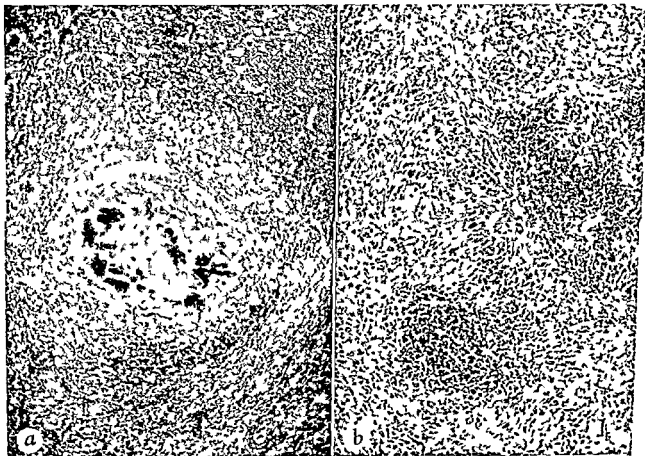
FIG 25 2—Tuberculous changes in cervical lymph nodes of swine a Mammalian tubercle bacillus infection The peripheral fibrosis, necrosis and calcification are typical of lesions due to the bovine or the human type of tubercle bacilli Hematoxylin eosin X 40 b Avian tubercle bacillus infection Diffuse cellular proliferation with little necrosis Hematoxylin-eosin X 95
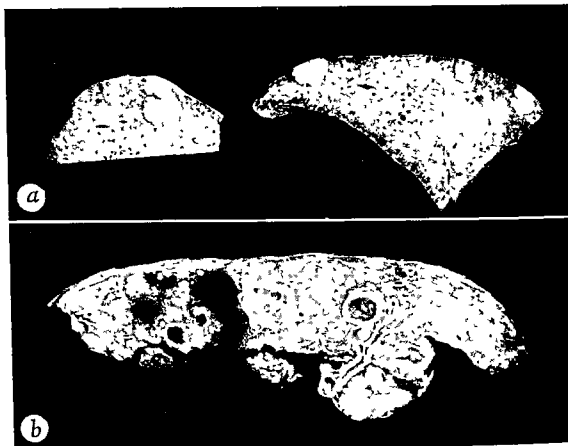
FIG. 25.3—*a*. Portions of liver from a case of generalized tuberculosis in a hog due to bovine tubercle bacilli. The nodular masses are well separated from the normal-appearing substance of the liver. *b*. Portion of lung from the same case. Here also the lesions occur as round, distinct nodules. (Photographs furnished by Dr. W. H. Feldman.)

an unequivocal diagnosis can be made only on the basis of bacteriologic procedures designed for the isolation, identification, and typing of the tubercle bacillus.[a]

SUMMARY AND RECOMMENDATIONS

The occurrence of tuberculosis in swine is related to the prevalence of this disease in other species. In the United States, the avian tubercle bacillus is the most common cause of tuberculous infection in swine. Although the incidence of tuberculosis among swine has been decreasing in this country since the 1930's, the loss is still considerable. At present, in abattoirs under federal supervision, the percentage of animals found to have tuberculous lesions is about 180 times greater for swine

than for cattle. To control tuberculosis in swine, efforts must be directed toward eliminating tuberculosis in fowl.

In the vast majority of cases, the lesions of tuberculosis in swine, as detected by the meat inspection services, are limited to small foci in a few lymph nodes of the alimentary tract. In a relatively high percentage of such lesions, bacteriologic studies have failed to reveal tubercle bacilli. The data on incidence of tuberculosis in swine are unreliable to the extent that some of the necrotic, granulomatous processes in lymph nodes of swine may not actually be tuberculosis.

A fruitful area for research is presented by the lack of information on the causes of localized necrotic lymphadenitis in swine. It is desirable to determine what characteristics, if any, may be used grossly to differentiate true tuberculous lesions

[a] Methods for identification of the type of tubercle bacilli may be found in the monograph by Feldman (1938a).

from other morbid changes. The relationship of *C. equi* to tuberculosis-like lesions in swine should be investigated.

Symptoms and signs of tuberculosis in swine are evident when the infection is disseminated and causes malfunction of various organs and massive enlargement of lymph nodes. However, these changes are not sufficiently characteristic to be of diagnostic value.

The appearance at necropsy of generalized tuberculous disease in swine plus the demonstration of acid-fast bacilli in the lesions is a fairly reliable diagnostic criterion. However, an unequivocal diagnosis is possible only on identification of tubercle bacilli by appropriate bacteriologic procedures.

For the intradermal tuberculin test in swine, both avian and mammalian tuberculin must be used. A positive reaction to one tuberculin or to the other will indicate, in most cases, the type of infecting tubercle bacillus. At present, no standard procedure is recommended for applying and for interpreting the tuberculin test in swine. Precise information is lacking with respect to the most suitable site of injection, the amount and type of tuberculin, and the optimal period for observing the results. The diagnostic value of the tuberculin test in swine will be on a firmer basis when investigations are made to determine the specificity and limitations of the test.

The eradication of tuberculosis in swine as well as in other species is dependent on the availability of an economical and specific means of detecting infected animals. It is suggested that studies be initiated to explore the possibility of developing a serologic test for tuberculosis. Such a test would require only one handling of the animal. Furthermore, a routine serologic test for tuberculosis could be performed on blood specimens submitted for other tests, such as tests of brucellosis.

REFERENCES

ALBISTON, H. E., PULLAR, E. M., AND GRAYSON, A. R.: 1954. The epidemiology of tuberculosis in Victorian pigs. Australian Vet. Jour. 30:561.

BANG, B.: 1899. *La lutte contre la tuberculose animale par la prophylaxie.* Nord. Med. Ark., No. 22, 32.1

BANG, O.: 1917. Undersøgelser over nogle Tuberkuliners Reaktionsevne. Kgl. Veterinaer- og Landbohøjskoles Aarsskrift, p. 335.

BANKIER, J. C.: 1916. Tuberculous lesions of swine. II. Survey of lesions found in prairie provinces, especially in Alberta. Canad. Jour. Comp. Med. 10.250.

BAUMANN, R., KRENN, E., AND LIEBISCH, H.: 1955. Die käsige Lymphknotenentzündung der Schweine. 3. Mitteilung. Übertragungsversuche Wien tierärztl. Monatschr. 42.209

——————. 1956. Über die käsige Lymphknotenentzündung der Schweine. 5. Mitteilung. Tierversuche mit Reinkulturen. Wien tierärztl. Monatschr. 43.341

BENDIXEN, H. C.: 1950. Bovine tuberculosis. *In:* The Fight Against Tuberculosis in Denmark. National Association for the Fight Against Tuberculosis. Copenhagen, NYT Nordisk Forlag Arnold Busck, p. 75

——————. 1956. Personal communication

——————, AND JEPSEN, A.: 1938. *Corynebact. equi* (Magnusson, 1923) som Aarsag til tuberkulose lignende Suppurationsprocesser hos Svin, navnlig i Halslymfekirtler. Medl. dansk Dyrl. 21.401.

BUTLER, W. J., AND MARSH, H.: 1927. Tuberculosis of human type in garbage fed hogs. Jour. Amer. Vet. Med. Assn. 70.786.

CLAPP, K. H. 1956. Tuberculosis like lesions in swine in South Australia. Australian Vet. Jour. 32.110.

COMMÉNY, H.: 1953. Statistiques comparées de morbidité tuberculeuse bovine et porcine aux abattoirs du Havre de 1932 à 1952. Recueil méd vét. 129.505.

CORNELL, R. L., AND GRIFFITH, A. S.: 1930. Types of tubercle bacilli in swine tuberculosis. Jour. Comp. Path. and Therap. 43.56

COTCHIN, E.: 1940a. Tubercle bacilli in lesions of the submaxillary lymph nodes of swine. Jour. Comp. Path. and Therap. 53.310.

——————. 1940b. *Corynebacterium equi* in the submaxillary lymph nodes of swine. Jour. Comp. and Therap. 53.298.

CRAWFORD, A. B.: 1938. Studies in avian tuberculosis. I. Avian tubercle bacilli in generalized disease in swine. Amer. Rev. Tuberc. 37.579.

ELIAN, A., EL AFFIFY, A., AND ZAKI, O. A.: 1953. The incidence and typing of tuberculosis in swine in Egypt. Jour. Egypt. Med. Assn. 36.723.

FELDMAN, W H 1936 The recovery of virulent tubercle bacilli from the tissues of swine intended for food Jour Infect Dis 59 43

—— 1938a Avian Tuberculosis Infections The Williams and Wilkins Co Baltimore

—— 1938b Generalized tuberculosis of swine due to avian tubercle bacilli Jour Amer Vet. Med Assn 92 681

—— 1939 Types of tubercle bacilli in lesions of garbage fed swine Amer Jour Pub Health 29 1231

—— 1952 Tuberculosis In Diseases of Poultry, 3rd ed Editors, H E Biester and L H Schwarte Iowa State College Press Ames Iowa p 341

—— 1955 Tuberculosis In Diseases Transmitted From Animals to Man 4th ed Editor, T G Hull Charles C Thomas Springfield Ill p 5

——, AND KARLSON, A G 1940 Avian tubercle bacilli in tonsils of swine. Jour Amer Vet Med Assn 96 146

—— Moses, H E. AND KARLSON A. G 1940 Corynebacterium equi as a possible cause of tuberculous like lesions of swine Cornell Vet 30 465

FOURIE, P J J DE WET, G J, AND VAN DRIMMELEN, G C 1950 Tuberculosis in pigs caused by M Tuberculosis var hominis Jour South Afr Vet Med Assn 21 70

GIRARD G 1948 Prémunition antituberculeuse des porcins par le B C G à Madagascar In Premier Congrès International du B C G Institut Pasteur, Paris, p 161

GRAHAM, R AND TUNNICLIFF, E A 1926 Fowl tuberculosis in swine Trans Ill State Acad Sci 19 138

HAYES, F M, HARING, C M AND TRAUM, J 1932 Vaccination of swine against tuberculosis with Calmette Guérin culture B C G Hilgardia 7 235

HOLTH R AND AMUNDSEN, H 1936 Fortsatte undersøkelser over bacíltypene ved tuberkulose hos svinet på Østlundet. Norsk Vet Tdskr 48 2

HUBNER, K 1956 Tuberkulosebekampfung im Spiegel der Fleischbeschaustatistik Auswertung der Fleischbeschaustatistik 1950-54 Die Fleischwirtschaft 8 122

JUNDELL, I, AND MAGNUSSON, H 1931 Recherches expérimentales relatives à l action du BCG sur le porc. Ann Inst Pasteur 47 403

KARLSON, A G AND FELDMAN, W H 1940 Studies on an acid fast bacterium frequently present in tonsillar tissue of swine Jour Bact 39 461

—— Moses H E. AND FELDMAN, W H 1940 Corynebacterium equi (Magnusson 1923) in the submaxillary lymph nodes of swine Jour Infect Dis 67 243

LANZ, ERWIN 1955 Über die Tuberkulose und die intrakutane Tuberkulinisierung beim Schwein Schweiz Arch f Tierheilk 97 229

LEE, C D 1956 A survey of avian swine tuberculosis and methods for its control Proc. 92nd Ann Meet Amer Vet Med Assn p 420

LUKE, D 1931 Studies in tuberculous sensitivity in the pig III The tuberculin skin reaction in the sow Vet Rec. 64 344

—— 1953 The intradermal tuberculin test in the pig Vet. Rec. 65 533

McCARTER, JANET, BEACH, B A AND HASTINGS E G 1935 The relation of the avian tubercle bacillus to tuberculosis in swine and incidentally in cattle Jour Amer Vet Med Assn 86 168

McDIARMID A 1956 Tuberculin testing of pigs Vet Rec 68 298

MITCHELL, C A WALKER R V L AND HUMPHREYS F A 1934 Types of tubercle bacilli found in swine of two accredited areas Rep of Vet Director General, Department of Agr Canada p 43

MOHLER, J R AND WASHBURN, H J 1907 Tuberculosis of hogs Its cause and suppression U.S.D.A 24th Ann Rep Bur Anim Ind p 215

OTTOSEN, H E 1915 Undersøgelser over Corynebacterium Magnusson Holth specielt med Henblik paa dens serologiske Forhold A/S Carl Fr Mortensen Copenhagen

PALLASKE, G 1931 Studien zum Ablauf zur Pathogenese und pathologischen Anatomie der Tuberkulose des Schweines (Beitrag zum vergleichenden Studium der Tiertuberkulose) Zeitschr f Infektionskrankh, parasit krankh u Hyg d Haustiere 39 211

PALS, C H 1957 Personal communication

PATERSON, A B 1949 Tuberculosis in animals other than cattle III Vet Rec. 61 880

PICKARD, J R 1952 Tuberculosis condemnations Proc. 56th Ann Meet U S Livestock San. Assn p 141

PLUM, N 1946 Om Vaerdien af den makroskopiske Diagnose af de Holthske Processer Maandsskr Dyrl 30 97

—— AND SLYNGBORG, N C 1938 Smittefarlig Tuberkulose (specielt Lungetuberkulose) hos Svin Maandsskr Dyrl 50 473

PULLAR E. M AND RESHBORN B H 1954 The accuracy of the avian intradermal tuberculin test in pigs Australian Vet Jour 30 221

PULLIN, J W 1946 Tuberculosis lesions of swine 1 Survey of lesions found in Eastern Canada Canad Jour Comp Med 10 159

RANNEY, A. F 1955 Status of Federal State Cooperative tuberculosis eradication Proc. 59th Ann Meet U S Livestock San Assn p 203

ROBINSON, E. M 1955 Tuberculosis of pigs Jour South Afr Vet Med Assn 26 259

SANZ, B   1930 Vaccination des bovidés avec le B C G au Chili Recueil med vet *106* 136

SCHALK, A I   RODERICK, L M , FOUST, H L., AND HARSHFIELD, G S   1935 Avian tuberculosis Collected studies No Dak Agr Exp Sta Tech Bull No 279

SCHROEDER, E C, AND MOHLER, J R   1906 The tuberculin test of hogs and some methods of their infection with tuberculosis U.S D A., Bur Anim Ind Bull 88

SVANDBERG, V   1935 Bakterientypen bei der Tuberkulose des Schweines in Finnland Arch wiss prakt Tierheilk 69 138

TAMMEMAGI L   1953 Tuberculosis like lesions in the submaxillary lymph nodes of pigs in Queensland Queensland Jour Agr Sci 10 81

THORNTON, H   1949 Text book of meat inspection   including fish, poultry and game Baillière Tindall and Cox London

TOPACIO, T   1933 Tuberculosis of swine in the Philippine Islands Philippine Jour Sci 52 349

U.S D A   1946 Summary of activities Agr Res Serv Meat Inspection Branch, Washington D C

VAN ES, L   1925 Tuberculosis of swine Univ Nebr Agr Exp Sta Circ. 25

———, AND MARTIN, H M   1925 An inquiry into the cause of the increase of tuberculosis of swine Univ Nebr Agr Exp Sta Res Bull 30

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Mycotic Infections

DERMATOPHYTES

Ringworm

Cases of ringworm in swine are apparently rare. In excellent articles on ringworm in animals, Georg (1954) and Blank (1953) make no reference to the disease in swine. Kinsley (1936), Anthony (1947), and Glässer *et al.* (1950), all indicate that ringworm is a rare condition in swine. Mc-Pherson (1956) quotes Ainsworth and Austwick as being unable to find dermatophytes in porcine samples in their survey of animal dermatophytes in Great Britain.

ETIOLOGY

McPherson (1956) has reported the only case of porcine ringworm backed by cultural studies that the author has found. He recovered *Trichophyton mentagrophytes* from lesions on a Large White pig in Scotland.

The cause of ringworm is listed by the other authors above as *Trichophyton tonsurans*, and in addition Anthony lists *Microsporum audouini*. Kral (1955) does not identify the species listing "Trichophyton sp." as the cause of porcine ringworm. *T. tonsurans* is generally regarded in this country as a pathogen of humans, so it can probably be assumed that in the United States the cause of ringworm of swine has not been positively identified.

CLINICAL SIGNS

The appearance of this condition in swine is much the same as in horses and cattle. The lesions are seen primarily on the back, sides, and head. Some authors also list lesions on the lower abdomen and inner surface of the thigh. The lesions begin as small, raised, grayish papules. These become covered with reddish scabs. At the same time, an inflammatory response causes the area to become thickened and red. The size of the lesion progresses outward on all sides of the peripheral inflammatory region, forming the typical "ringworm." Meanwhile, the center of the lesion has tended to heal, leaving an area in which some of the hairs have disappeared or broken off, with bristles and some normal-appearing hair remaining (Fig. 26.1). The area may contain scales of

FIG. 26.1 — Ringworm lesion on neck and jowl of pig. The dark areas are the result of biopsies. (Courtesy Prof. E. A. McPherson, Royal [Dick] School of Veterinary Studies, Edinburgh.)

dandruff like material Healing may not be complete in the central portion of the lesion, and irregular red areas may remain The lesions differ from those of cattle primarily in that they are red rather than grayish to yellowish, and do not present an encrusted appearance The lesions in swine usually do not cause itching although McPherson's cases did Affected areas may become 5 inches (127 mm) in diameter

DIAGNOSIS

A positive diagnosis is attained by demonstration of the spores of the fungus in selected hairs or *skin scrapings* from the lesion Cultural identification of the fungus is a practical procedure for those having access to a laboratory offering this service However, the gross appearance of the lesion is so typical as to make field diagnosis quite reliable

The spores on the hairs in swine ringworm are often difficult to demonstrate microscopically For this purpose, hairs at the edge of the lesion should be selected In addition, a deep skin scraping in the same region should be made with a curette or sharp, curved belly, scalpel blade (Bard Parker No 22)

The hairs can be examined under a cover glass in a drop of mineral oil using the low power magnification (100 X) for scanning and high dry for detailed examination The use of reduced light as obtained by racking down the condenser will facilitate finding the spores The use of *Amann's medium (lactophenol cotton blue)* serves as a combined fixing agent, stain, and mounting fluid It has the following formula

Phenol crystals	20 gm
Lactic acid, syrup	20 gm
Glycerol	40 gm
Water	20 ml

Dissolve the above together with gentle warming in a hot water bath and add

Cotton blue	0 05 gm.

The mycelia and spores stain blue *Trichophylon* spp have spores either within or surrounding the hair follicle Most animal Trichophytons are of the ectothrix

type in which the spores are arranged along (outside) the shaft of the hair (Fig 26 2) They appear as refractile bodies under the microscope Naturally occurring oil droplets can be confused with them and it may be desirable to extract the specimen with ether or chloroform to remove the oil droplets Pigment granules may also be confusing and may be bleached out with dilute hydrogen peroxide

Examination of the deep skin scrapings can be facilitated by dissolving the epithelial debris in hot (do not boil) 10 per cent sodium or potassium hydroxide

Trichophyton spp do not fluoresce under the ultraviolet (Wood's) lamp, so this technique, which is helpful in detecting *Microsporum canis*, is of no value in ringworm of swine

TREATMENT

Clipping of the hair around the lesion and removal of any encrustations are indicated preliminary steps in the treatment of ringworm lesions Kral (1955) has recently reviewed the treatment of animal dermatomycoses He recommends general measures such as improvement of diet and sanitation Systemic use of iodides such as sodium iodide, given orally or intravenously, is recommended as good supportive therapy, especially in severe fungous infections Kral uses newly developed iodine preparations in which the iodine is combined with certain types of synthetic detergents such as ethylene oxide condensates of propylene glycol Three to four applications at 2 day intervals have been sufficient to effect a cure in 12 to 20 days in cases treated by him in horses cattle, dogs, cats and monkeys Ten per cent Clorox rubbed in well with a tooth brush has also been recommended for cattle ringworm by several authors Captan[1] (N trichloromethyl - 1 - cyclohexane 1 2-dicarboximide) and Phemerol[1] 1 500 have also been recommended for bovine ringworm by Hoerlein (1956) and Fox (1956) and might be tried in the disease in swine
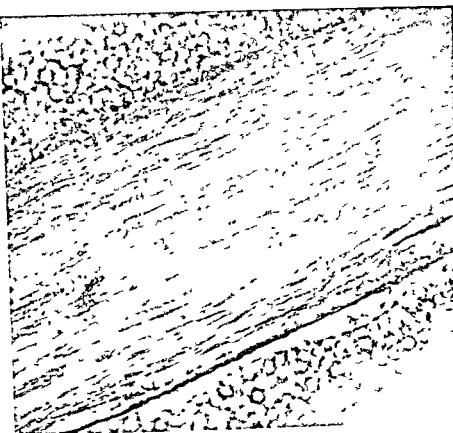
FIG. 26.2—Spores of ecto-thrix type along guinea pig hair, Trichophyton mentagrophytes. X 475. (Courtesy Dr. Lucille Georg, U.S.P.H.)

IMMUNITY

There is some evidence (Kral, 1953) of at least a local immunity developing at the site of a previous ringworm infection of animals. In speaking of humans, Beamer (1955) states,

In general, the serums of patients with dermatomycoses contain no demonstrable antibodies, but Wharton and his associates have demonstrated precipitans in the serums of immunized rabbits. Failure to demonstrate circulating antibodies in patients may mean that none are formed in such superficial infections, or the antigens, which are extremely difficult to prepare and standardize properly, may not be sufficiently sensitive

EPIZOOTIOLOGY AND CONTROL

Glasser et al. (1950) quote Schindelka as indicating that ringworm in cattle will spread to swine. These same authors state that Magnussen artificially infected a pig with ringworm fungi obtained from a horse.

The disease spreads readily between pigs once the condition is established in those animals. Affected animals should be isolated as soon as detected and until the lesion has completely healed. Rubbing-posts or trees used by the pigs should be thoroughly scrubbed with strong soap and, when dry, painted with tincture of iodine.

Pityriasis Rosea

This is a ringworm-like condition of pigs resembling pityriasis rosea of man The cause of the condition in man or animals is unknown (Kral, 1953). A description of the disease is included here for comparison with ringworm.

Clinical signs. This is primarily a disease of young pigs. It may be seen in individual animals or in whole litters. At the onset of the condition digestive disturbances such as inappetence, vomition, and diarrhea may be seen. After the appearance of the skin lesions these symptoms cease. The lesions begin as pea sized, slightly thickened red spots which often coalesce to form nodules. The spots are depressed in the center and covered with a brown dandruff and scab. This material is soon lost from the center of the lesion but remains at the periphery which enlarges. These raised peripheral swellings form tortuous

FIG. 26.3 — Young pig affected with Pityriasis rosea, a condition clinically resembling ringworm. (Courtesy Prof. E. A. McPherson, Royal [Dick] School of Veterinary Studies, Edinburgh.)

red-to-blue strips one half to one cm. broad that may cover large areas of the body. The hair is not completely removed by this disease process (Fig. 26.3).

Treatment. Glasser et al. (1950) indicate that the condition heals spontaneously in 2 or 3 weeks. These authors also quote Grimm as successfully using a salve consisting of salicylic acid 5 gm., sulphur sublimate 15 gm., and vaseline 125 gm.

SYSTEMIC MYCOSES

With the exception of actinomycosis, it does not appear that the systemic mycoses are of economic importance in this country. References on the subject from abroad also are meager. The most amazing thing about systemic mycotic diseases of swine is that they are so rare. In exhaustive review-articles by Saunders (1918) and Beneke (1953) the former noted only 6 reports of

systemic mycoses, 5 of which were from abroad. Beneke added no cases not reported by Saunders. The proceedings of a conference on histoplasmosis (1952) contains no reference to the disease in swine. Due to the rooting habits of swine, it would seem that these animals would have ample opportunity to become infected in endemic areas.

Actinomycosis

References on actinomycosis of swine are not difficult to find in the veterinary literature; however, cultural identification is usually lacking. Vawter (1946) states, "In spite of the many reports intimating that *A. bovis*, or a variant thereof, occurs in the actinomycotic lesions of swine, search of many papers has not revealed any detailed cultural description of the swine type other than that given by Magnussen." The clinical diagnosis of actinomycosis is placed on many fibrous enlargements of swine, especially those of the udder, that are in fact caused by *Actinobacillus lignieresi*, *Micrococcus* sp. (botryomycosis) or *Corynebacterium pyogenes*.

Etiology. True actinomycosis is caused by *Actinomyces bovis*. There is a popular misconception that this organism is found on grass or awns. Actually, it is a saprophyte of the oral cavity and, when found on vegetable particles in lesions, it has gained access to the tissues by mechanical transference (Skinner et al., 1917). True cases of actinomycosis of the udder of swine are rare and probably caused by tooth wounds by nursing pigs, or by mechanical injury by straw, etc., contaminated with saliva containing *Actinomyces bovis*. The occurrence of lesions in the spleen, liver, and kidney suggests the possibility of systemic infection of the udder. All of these sites probably become infected by metastasis from the digestive tract or lungs.

The organism is anaerobic or microaerophilic and difficult to isolate and maintain in culture. Pus or a granule from a lesion can be used for culture. The material can be plated on veal liver serum agar plates and inoculated into deep agar shake cul-

tures as recommended by Vawter (1946).
Skinner et al. (1947) recommend veal in-
fusion agar pH 7.4 containing 1 per cent
glucose as the most satisfactory medium
for primary isolation. Vawter (1946) also
recommends serumized media, deep col
umns of glucose veal-liver broth containing
0.05 per cent agar and cooked meat me-
dium with sodium thioglycollate. This fun-
gus will not grow on Sabouraud's medium.
Cultures should be transferred every 14 to
21 days. If kept at 5° C. in buffered cooked-
meat medium they can be transferred every
2 months.

Acid without gas was formed by Vawter's
(1946) swine strain in glucose, levulose,
galactose, maltose, lactose, sucrose, treha
lose, raffinose, dextrin, inositol, salicin,
glycerol, and inulin. The reaction was de-
layed 7 days in the case of inulin, inositol,
raffinose, and salicin, and 14 days for glyc-
erol. Substances not fermented were arab-
inose, mannite, dulcitol, xylose, rhamnose,
and sorbitol. Fermentation was determined
by withdrawing a small amount from the
tube and testing with bromthymol blue on
a porcelain plate.

The organism is very pleomorphic, vary-
ing from long, branching hyphal forms to
rods of various sizes — diphtheroid, hyphal,

or coccus forms. These are usually Gram-
positive but may have a beaded appear-
ance. They are not acid fast.

The pus from actinomycotic lesions usu-
ally contains granules that are yellow-to-
brown calcareous structures. These are the
so called "sulphur granules," which are of
diagnostic importance (see Diagnosis).
These radiating clublike structures stain
Gram-negative.

Clinical signs. This chronic disease in
swine usually involves the soft tissues, and
as Vawter (1946) indicates, ". . . tends to
follow a pattern of tissue localization some-
what analogous to actinomycosis in man,
wherein pulmonary, adenocervical, or skin
lesions may appear."

Internal lesions in swine will result in
slow weight gains or weight losses that may
or may not be noticed by the owner. Should
the organism localize in the udder or skin,
a gray, hard, granulation tissue will be
formed around the lesion, which will be
easily detected. Such lesions of the udder
attain considerable size, becoming pendu-
lous and sometimes hanging to the ground
(Fig. 26.4).

Pathological changes. Microscopically,
Smith (1953) describes the lesions as con-
sisting of the ray fungus in the center, sur-

FIG. 26.4 — Actinomycotic-
type lesions of udder of a
sow. Most lesions of this
nature are caused by Mi-
crococcus spp. (botryomy-
cosis), Actinobacillus spp.,
or Corynebacterium spp.,
rather than Actinomyces
bovis. (Courtesy Dr. E. R.
Frank, Kansas State Col-
lege.)

rounded by a zone of pus containing living neutrophils. Next is a zone of endothelioid granulation tissue. Lymphocytes and scattered Langhans' giant cells may be in this area. A fibrous capsule surrounds the endothelioid zone. Calcification of the rosettes (ray fungi) may occur, but caseation is not seen. The granuloma may be made up of a confluence of such structures.

Diagnosis. Differentiation of the causes of these lesions is probably only of academic interest to the practitioner. He will be able to distinguish between uncomplicated cases of actinomycosis, actinobacillosis, and botryomycosis (micrococcosis [staphylococcosis]) infection on the basis of morphology and staining characteristics of organisms found in smears. Actinomycosis is caused by Gram-positive organisms while actinobacillosis organisms are Gram-negative. The micrococci causing botryomycosis are Gram-positive and have the characteristic coccoid and often "grape cluster" appearance.

All of these diseases on occasion will produce granules in the pus. These are helpful in differential diagnosis. To aid in finding them, the pus can be diluted with 10 per cent potassium hydroxide and poured into a Petri dish, or as Hagan (1943) recommends, the pus can be placed in a tube of broth or saline and shaken to dissolve the mucin holding the material together. Then it is poured into a dish, and the granules, which do not dissolve, are selected. These are crushed and used for culture or staining.

Treatment. In surgically accessible areas, removal of the granuloma, together with intensive treatment with parenteral penicillin-streptomycin and intravenous sodium iodide, is recommended. Only in the case of valuable breeding animals will treatment be economically feasible.

Immunity. Mathieson *et al.* (1935) have suggested that actinomycosis does not result from the first invasion by the causative organism, but by repeated exposures which sensitize the host to actinomyces. In this way it would be similar to *infection with Coccidioides immitis.* In swine there is little

opportunity to determine if immunity develops, as these animals seldom have a chance to acquire a second infection.

Magnusson (1928) found three serological types. Type A was characteristic of cattle, types B and C of swine.

Epizootiology and control. The infection caused by *A. bovis* is not one that usually becomes a herd problem in swine. Vawter's (1946) case involving 2 or more affected animals on the same farm, each of 2 successive years, is the only case refuting this generalization that has come to the author's attention.

Apparently this is an infection arising from the accidental invasion of the body by organisms that exist normally in the mouth and tonsillar region.

Pus from animals affected with *Actinobacillus lignieresi, Micrococcus aureus* or *Corynebacterium pyogenes* is infectious. Due to the difficulty in differentiating clinically between the lesions caused by these organisms and actinomycosis, which is relatively noncontagious, it is highly recommended that pus from any abscess not be allowed to contaminate the premises or equipment.

The condition called "throat or cervical abscesses" caused by Lancefield group E streptococci might well be added to the above list and handled in the same manner.

Mucormycosis

Mucormycosis has received more notice than any of the systemic mycotic infections except actinomycosis, yet only 11 cases have been found in the literature. Nine of these were reported by Christiansen (1929) and Nielsen (1929), one by Davis *et al.* (1955), and one by Vink (1941). Saunders (1948) objects to the cases described by Christiansen and Nielsen on the grounds that they describe septate mycelia in a genera that is by definition non-septate. However, Gleiser (1953) quotes Emmons as being of the opinion that mucor may become septate under certain conditions.

In an earlier report, Christiansen (1922) described 2 swine cases (also included in

the 1929 report) that occurred at an abattoir within a few months of each other These pigs had in the abdominal cavities, primary mycotic granulomas, which had metastasized to the lungs in one case and the liver in the other The nodules had a caseous center surrounded by epithelioid tissue containing giant cells and plasma cells This area, in turn, was surrounded by a dense connective tissue capsule The affected areas were permeated with hyphae and eosinophiles One pig had been moribund for 8 days prior to slaughter, whereas the other had been normal The molds recovered from these lesions were identified as *Rhizopus equinus* (changed to *R suis* in the 1929 report) and *Absidia ramosa* These were pathogenic for rabbits, rats, and mice by intravenous and intraperitoneal injection A macerated lung nodule and a pure culture of Rhizopus were inoculated into some pigs with negative results

The seven cases reported later by Christiansen (1929) and Nielsen (1929) were caused by Absidia and produced nodules in the small intestine and mesenteric lymph nodes with occasional metastases The caustive mold was easily recovered, and the histological structure of the nodules was similar to that described above

Saunders (1948) refers to a case where *Absidia corymbifera* was recovered from the submaxillary lymph node of a pig

Mucoraceae are such common air contaminants that histological evidence in affected tissues should be found to support cultural evidence before making a diagnosis of mucormycosis Zimmerman (1957) has commented on the importance of his topathological examination in fungus diseases

Cryptococcosis

A report by Vuillemin (1901) reports the recovery of *Cryptococcus granuloma togenes* from a granuloma in the lung of a pig This was probably *C neoformans*

Epizootic lymphangitis. (*Zymonema farciminosa, Histoplasma farciminosum,*

Cryptococcus farciminosus) Oehl (1929) has described a case of *lymphangitis epizootica* in a pig which he attributed to *Cryptococcus farciminosus* His description of the causative agent does not permit confirmation A yearling boar was affected in the skin of the right front leg, the head, side, flank, and both hind legs The right front leg was twice normal size Numerous nodules were seen, some fistulated and connected with each other by enlarged lymphatics The regional lymph nodes contained small purulent foci The lungs had numerous pea to bean sized abscesses that contained a yellow, creamy pus There were no infected horses on the farm, and the source of the infection was not traced

Moniliasis (Candida, Oidium, Thrush)

Kovalev (1947) reported moniliasis in young pigs that were affected in the nose, gums, and lips, with swelling of the upper jaw and accompanied by a foul odor The causative agent was identified as *Oidium albicans* (*Monilia albicans*) The organism is said to produce a systemic toxin in addition to local necrosis Affected pigs became emaciated rapidly and died

Quin (1952) described cases of alimentary mycosis, involving areas from the esophagus on through the digestive tract, that appeared in pigs on excessive or prolonged intake of antibiotic residues The clinical picture varied, some droves developing persistent scouring, encephalitic symptoms, and excessive thirst, with a variable death loss At necropsy these pigs had pigmented patches on the mucosa of the digestive tract resembling pseudomembranes that were 1–2 mm thick, mostly in the small intestine In one drove, a pig was seen in which the entire intestinal mucosa was hidden by a gray-colored mold growth A pure culture of Monilia was recovered from a piled up inflammatory fungus lesion that occluded the esophageal-gastric junction When the pseudomembrane peeled off, the underlying tissue varied from red to black in color

Further trouble and losses ceased when

antibiotic intake was stopped in these herds

Quin (1957) has also recommended a treatment consisting of a solution of 1 lb copper sulfate in 1 gal water, of which so lution one pint is mixed with each 25 gal drinking water for 6 to 8 days

Aspergillosis

Saunders (1948) quotes Wyssman as re porting a case of aspergillosis in a pig, pre sumably located in the lungs

Spindler and Zimmerman (1945) have reported the recovery of an unidentified species of Aspergillus from ground up sar cocysts (Miescher s sacs) Confirmation of this work is lacking

Maddy (1956) quotes Thornton as pointing out that in the United States 90 per cent of garbage-fed hogs have sarco cysts, whereas only 15 per cent of others are affected

Sarcocysts are often visible macroscop ically, especially if calcified, however, they are usually microscopic in size They are present most frequently in the abdominal and diaphragmatic muscles of swine In festation may be severe enough to cause condemnation of an entire carcass

Nocardia

Emdin (1954) reported the isolation of an "asteroides" sp from granulomatous lesions of the lungs, kidneys, liver, and spleen of an 8 month-old barrow, in good condition, that was presented for routine slaughter The lesions were cultured and material in jected into guinea pigs Lesions of the lung and spleen, similar to those of the hog, were produced The fungus was reisolated from these guinea pig lesions Nocardia are acid fast, aerobic, easily cultivatable or ganisms that are pathogenic for man Great care should be taken not to inhale spores from uncovered Petri dish cultures

Cultures can be handled more safely if carefully flooded with sterile mineral oil prior to transfer

General Considerations

Granulomatous lesions caused by fungi are rare and will be found most often at necropsy The question facing the prac titioner finding internal lesions will be, Is this tuberculosis? If desired, he can make a smear from material at the edge of the caseous center and stain with Kinyoun s acid fast stain, which has the advantage of not requiring heat as does the Ziehl Neel sen stain The formula is as follows

Basic fuchsin	4 gm
Phenol crystals	8 gm
Alcohol (95 per cent)	20 ml
Distilled water	100 ml

Smear on a clean slide the material to be examined and apply Kinyoun s stain for three minutes Wash with water and de colorize with acid alcohol (2 ml conc HCl in 98 ml of 95 per cent alcohol) un til no more color comes out Wash in water and counterstain with 1 per cent aqueous methylene blue Wash with water, dry, and examine under oil immersion for red (acid fast) organisms, others will be blue Hy phal elements may or may not be seen in these smears if a fungous disease is pres ent If the search for acid fast organisms is negative, a portion of the lesion (includ ing all parts from the caseous center to the normal tissue) should be placed in 10 vol umes of 10 per cent formalin and sent to a laboratory for histopathological examina tion, with the request that routine, acid fast and fungous stains be employed An other portion should be sealed in a plastic (deep freeze) bag and placed in cracked ice or dry ice for shipment to a laboratory for cultural examination The history ac companying the specimens should indicate the nature of the lesions and suspected clin ical diagnoses

REFERENCES

ANTHONY, D J 1947 Diseases of the Pig and Its Husbandry, 2nd ed Williams and Wilkins Co, Baltimore

BARRON, C N 1955 Cryptococcosis in animals Jour Amer Vet Med Assn 127 125

BEAMER, P R 1955 Immunology of mycotic infections Amer Jour Clin Path 25 66

BENEKE, E S 1953 Detection of mycotic infection in animals Mich St Coll Vet 13 219

BENHAM, RHODA W 1955 The genus Cryptococcus The present status and criteria for the species Trans N Y Acad Sci Ser II 17 418

BLANK, F 1955 Dermatophytes of animal origin transmissible to man Amer Jour Med Sci 229 302

CHRISTIANSEN, M 1922 Deux cas de mycose généralisée chez le porc, determinés par de mucor inées Compt rend Soc Biol Paris 86 461

———— 1929 Mucormykose beim Schwein Virchows Arch f Path Anat 273 829 (Quoted by Saunders, 1918)

DAVIS, C. L., ANDERSON, W A AND McCRORY, B R 1955 Mucormycosis in food producing animals Jour Amer Vet Med Assn 126 261

EMDIN, R 1954 Micosi generalizyata in suino Ann Fac Med Vet Pisa 7 59

FOX, F. H 1956 In Diseases of Cattle American Veterinary Publications Inc Evanston, Ill

GEORG, LUCILLE K 1954 The diagnosis of ringworm in animals Vet Med 49 157

GLÄSSER, K, HUPKA, E, AND WETZEL, R 1950 Die Krankheiten des Schweines, 5th ed M & H Schaper, Hannover, Germany

GLEISER, C A 1953 Mucormycosis in animals Jour Amer Vet Med Assn 123 441

HAGAN, W A 1913 The Infectious Diseases of Domestic Animals Comstock Publ Co Inc. Ithaca, N Y

HOERLEIN A B 1956 In Diseases of Cattle American Veterinary Publications Inc Evanston Ill

KINSLEY, A T 1936 Swine Practise Alexander Eger Chicago

KOVALIV A A 1947 Mycotic aphthous stomatitis in suckling pigs Veterinariya 24 18 (From abstract in Jour Amer Vet Med Assn 1947 111 313)

KRAL, F 1953 Veterinary Dermatology J B Lippincott Co, Philadelphia

———— 1955 Classification, symptomatology and recent treatment of animal dermatomycoses (ringworm) Jour Amer Vet Med Assn 127 395

McPHERSON, E A 1956 Trichophyton mentagrophytes Natural infection in pigs Vet Rec 68 710

MADDY, K 1956 Sarcosporidiosis In Diseases of Cattle American Veterinary Publications Inc Evanston Ill

MAGNUSSEN, H 1928 The commonest forms of actinomycosis in domestic animals and their etiology Acta Path et Microbiol Scand 5 170

MATHIESON, D R HARRISON, R, HAMMOND C AND HENRICI, A T 1935 Allergic reactions of actinomycetes Amer Jour Hyg 21 405 Quoted in Diseases of Cattle, 1956, American Veterinary Publications, Inc, Evanston, Ill

NIELSEN, N 1929 Mucormykose beim Schwein Virchows Arch f Path Anat 273 859 (Quoted by Saunders, 1918)

OEHL 1929 Lymphangitis epizootica beim Schwein Deutsch tierarztl Wochenschr 37 39

PROC OF CONF ON HISTOLASMOSIS 1952 U S Dept of Health Education, and Welfare, Publ Hlth Monograph, No 39

QUIN, A H 1952 Newer problems in swine diseases—control and treatment Canad Jour Comp Med 16 265

———— 1957 Personal communication

SAUNDERS, L Z 1948 Systemic fungous infections in animals A review Cornell Vet 38 213

SKINNER, C E, EMMONS, C W, AND TSUCHIYA, H M 1947 Henrici's Molds Yeasts and Actinomycetes John Wiley and Sons, Inc, New York, p 371

SMITH, H A 1953 The granulomas General pathology notes Texas A & M College, College Station, Tex

SPINDLER, L A AND ZIMMERMAN, H E, JR 1945 The biological status of sarcocystis Jour Parasit, Suppl 31 13

VAWTER, L R 1916 Pulmonary actinomycosis in swine Jour Amer Vet. Med Assn 109 198

VINK H H 1941 Mucormycose bij een varken Tijdschr v Diergeneesk 68 312 (Quoted by Saunders 1918)

VUILLEMIN, P 1901 Les blastomycetes pathogènes Rev Gen Sci 12 732

ZIMMERMAN, L E 1957 Some contributions of the histopathological method to the study of fungus diseases Trans N Y Acad Sci, Ser II 19 358

PARASITIC INFECTIONS

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CHAPTER 27

External Parasites

In North America, the economically important external parasites of swine are relatively few in number. Lice and mange mites are the principal ectoparasites that affect hogs, though other arthropods have been reported as pests of swine.

Parasites that live on the body surface of their hosts are usually termed external parasites or ectoparasites. For most species this distinction is satisfactory, but in the case of mange mites which burrow beneath the skin surface and may spend a part of their life cycle internally, the term may be misleading. All external parasites of swine belong in the phylum ARTHROPODA. This is a very large phylum of the animal kingdom. The parasitic species of importance to domestic animals are either insects, ticks, or mites, although a few species of Crustacea act as intermediate hosts for some helminths.

The phylum Arthropoda contains those invertebrates that have jointed legs. The members of one class known as the INSECTA are characterized by a body divided into three parts: (1) a head which bears a pair of antennae, (2) a thorax with 3 pairs of legs and often 2 pairs of wings, and (3) an abdomen which usually has no appendages. This large class includes parasitic insects such as flies, fleas, mosquitoes, lice, and many other species not parasites of domestic animals.

Another large class of arthropods containing parasitic forms of importance to

swine are the ARACHNIDA. This group is distinguished by having a cephalothorax and abdomen, the former bearing 4 pairs of legs in the adult. No antennae are present. Parasites of importance to swine in this group are the mites and ticks.

LOSSES DUE TO ARTHROPOD PARASITES

Depending on the habits of the species concerned, symptoms and lesions due to ectoparasites are quite variable. Most of the important external parasites attacking swine are permanent parasites which live on the skin surface or just below it. By piercing the skin they feed on blood or tissue fluids. In the case of lice and mange mites, they are so dependent upon their host that, if removed, they will die in a short time. In addition to those ectoparasites which are injurious because of their parasitic habits, there are those that may be carriers and transmit disease-producing organisms. Others cause sufficient irritation that secondary bacterial invasion occurs with resultant skin infections of various kinds. The majority of ectoparasites cause some degree of irritation to the skin surface. Infested animals spend a considerable part of their time attempting to alleviate the irritation by rubbing, scratching, and moving about restlessly. Feeding and resting periods are interrupted and, if the nutritional level of the animal is reduced, the resistance of the animal will be lowered and it may become more susceptible to

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other diseases As a result of the excessive irritation due to ectoparasites, accidental injuries may occur as the animal attempts mechanically to relieve the irritation

DIAGNOSIS AND CONTROL MEASURES

As a general rule, diseases of swine caused by arthropods are fairly easy to recognize In the case of the lice, ticks, and fleas, the parasites can usually be seen grossly Mange mites are not easily observed with the naked eye, the diagnosis should be confirmed by the use of a microscope for demonstration of the mites

The most important point in a diagnosis is the complete identification of the species concerned so that appropriate control measures may be recommended The life histories of arthropods vary to a great degree within species Effective control can be accomplished only if the habits and life history of the parasite are properly understood

The ideal method of ectoparasite control is complete eradication With the newer insecticides now available and with their high efficiency, control of many species is more easily accomplished than it was in the past. Control measures should aim at eradication or reduction of numbers and protection of the animal against further infestation

General sanitation of pens and yards is always of importance Proper drainage and manure disposal greatly aid in fly control Good husbandry and feeding practices will aid in minimizing the chances of parasitic infestation and in reducing losses due to parasites Such practices together with the use of appropriate insecticides, isolation of new animals entering a herd, and good management practices should eliminate losses and reduce damage due to ectoparasites

THE HOG LOUSE (Haematopinus suis)

The pig is unusual in that it is not the host of many different species of true insects (class INSECTA) Though fleas, flies, fly larvae, and mosquitoes are reported as infesting swine, the hog louse is the insect most commonly found on swine and in any locality where swine are raised It belongs to the order ANOPLURA, suborder Siphunculata, the members of which are equipped with sucking and piercing mouth parts

One species of louse, Haematopinus suis, (Fig 27 1) occurs on swine It is most frequently seen around the folds of skin of the neck and jowl, around the base and inside of the ears, on the inside of the legs, and on the flank This is the largest of the lice found on domestic animals In color it is grayish brown with brown and black markings The female is from 4 to 6 mm in length, the male being slightly smaller in size

According to Florence (1921), the eggs are laid one at a time and attached to the hog bristles by a clear cement When laid, the egg is a pearly white but gradually becomes more opaque and finally appears light amber in color A female may lay from 3 to 4 eggs per day, which hatch in
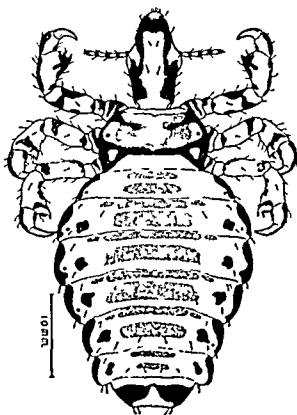
FIG 27 1 — Haematopinus suis, the hog louse (From Whitehead, 1942. Macdonald College Farm Bulletin No 7)

12 to 20 days, during her lifetime she may lay up to 90 ova, which are deposited over a period of 25 days After hatching in 12 to 20 days the nymphs undergo 3 moults, during which time they feed on the more tender parts of the pig's body such as the inside of the ear The life cycle from egg to egg is from 29 to 33 days and the aver age life span is about 35 days Like other lice, they do not leave their natural host and they cannot live off the host for more than 2 or 3 days When hungry, this species will feed on human blood if per mitted to do so

Since lice feed frequently, the continual puncturing of the skin to suck blood and lymph gives rise to considerable irritation In severe infestations the constant irrita tion and itching induces the pig to seek relief by scratching and rubbing vigorously against any available object This leads to skin laceration, bleeding, and a concentra tion of lice around the traumatized areas As the louse population increases, the hogs become restless, do not feed properly, be come unthrifty, and fail to make normal growth and weight gains A general lower ing of vitality and resistance make them more susceptible to attack by other para sites and contagious diseases It is generally considered that the hog louse is the trans mitting agent of swine pox virus Schang (1952) has observed, over a period of years in the Argentine, that swine pox does not appear without *Haematopinus suis* and that the disease is easily carried from af fected to unaffected pigs by this agent Since *H suis* is the only sucking louse of swine in North America, its presence on hogs will suffice for a satisfactory identifi cation

Control of the Hog Louse

Since the hog louse is a permanent para site and is spread by contact, control methods are directed toward louse de struction on the host.

Prior to the development and wide use of the chlorinated hydrocarbons many oily preparations were used for the control of this pest. Crude petroleum oil, crank case oil, and other processed oily materials were applied by hand, by the use of hog oilers or rubbing posts, and by means of medicated wallows Hand applications, rubbing posts, and hog oilers are useful for keeping louse infestations in check, but as a general rule they cannot be relied upon for the eradication of lice

The instinctive habit of the hog to wal low in water may be used to advantage in louse control By the use of oil in well constructed and maintained wallows, satis factory control may be accomplished with a minimum expenditure of money time and labor Kemper and Peterson (1955) give an excellent description of the con struction of hog wallows, their mainte nance and usage

Dipping of hogs is a most effective method of treatment but in many areas dipping vats are no longer available The widespread use of power sprayers on farms has introduced a convenient method of applying insecticides to animals and if done thoroughly, effective control can be ac complished Spraying equipment may be moved readily from place to place, which is easier than taking hogs to a central dip ping vat A disadvantage of the spraying method is the chance of not getting com plete coverage of the hogs in dipping complete coverage is assured. However if hogs are sprayed in small groups and con fined in a small pen with deep straw bed ding a thorough spraying can be carried out In addition to the complete coverage of the entire body, it is important that the inside of the ears be treated All animals in a herd *must* be treated

It is well to keep in mind that oil treated animals should be provided with shade or preferably, should be treated in late after noon or early evening If allowed to run in direct sun immediately after treatment, white skinned breeds especially, may be come scalded and blistered

The chlorinated hydrocarbons have be come widely used for control of hog lice When used as dips and sprays, Cobbett and Bushland (1956) state that benzene hexachloride (BHC) and lindane should

contain 0 06 per cent of the gamma isomer — the latter is the active insecticidal principle DDT should be used at a strength of 0 75 per cent whereas dips or sprays prepared with chlordane or toxaphene should contain 0 5 per cent of either chemical

These insecticides will kill all the lice on the hogs and in view of their residual action most of the young lice hatching after treatment will succumb However, there is always the chance that a few late hatching lice may survive the original dipping or spraying A second application 10 to 14 days after the original treatment is recommended

It also should be kept in mind that the chlorinated hydrocarbon insecticides are deposited to a variable degree in the tissues of animals to which they have been applied It is recommended that hogs should not be treated 30 days prior to slaughter Since there is always a possibility that hog lice or their eggs may have become separated from their host, infested premises should be cleaned and disinfected before clean hogs are admitted New animals being added to a herd should be quarantined and examined for lice, control measures should be taken if necessary

HOG MANGE

Mange in swine is a skin disease caused by mites Two types of mange affect pigs, one caused by *Sarcoptes scabiei* var *suis* which is a burrowing mite, the other is due to *Demodex phylloides* which inhabits the hair follicles or sebaceous glands Both species belong to the phylum ARTHROPODA, the class ARACHNIDA, and together with the ticks are placed in the order ACARINA The most common type of mange in the United States is sarcoptic. Demodectic, or follicular mange is less frequently seen

Sarcoptic Mange

The causative agent of sarcoptic acariasis is the mite known as *Sarcoptes scabiei* var *suis* This type of mange is widely distributed and is one of the troublesome

conditions with which swine growers have to contend It is most frequently encountered in parts of the country where the hog population is concentrated such as the Corn Belt area of the United States

Morphological differences between the species of *Sarcoptes* found on man and domestic animals are very slight Each is regarded as a variety of the form *Sarcoptes scabiei* Most of the varieties can be transferred from one host to another and the variety *suis* may establish in the skin of man Usually they live only for a limited time on unusual host animals, but during this time they may give rise to an annoying and a serious dermatitis It appears that there is a considerable degree of host specificity within this genus of mites, and probably biological races exist

Mange mites spend their entire lives on the host animal Sarcoptic mange mites are burrowing forms living in galleries or tunnels in the horny layers of the skin They are minute in size, roughly circular in outline, whitish gray in color, and scarcely visible to the naked eye They are about 0 5 mm in length The cuticle of the upper surface of the body is sculptured with fine wavy transverse folds or lines In the female (Fig 27 2) numerous, short, backward projecting spines may be seen on the dorsal surface

The mature mite has 4 pairs of short, stumpy, thick legs which are provided with sucker like organs at the tips of long unjointed pedicels on the first 2 pairs of legs in the female and the first, second and fourth pairs in the male (Fig 27 3) Other legs terminate in long bristles

The life history of this mite requires further detailed study Lapage (1956) states that the life cycle is not completely known However, it is probable that it is similar to that of *S scabiei* in man A new host probably is infested by an ovigerous female which penetrates the skin and works toward the horny layer (stratum corneum) of the skin Egglaying proceeds as the female burrows The eggs are oval in shape measuring 0 15 by 0 1 mm, and 2 or 3 are laid daily over a period of about a month
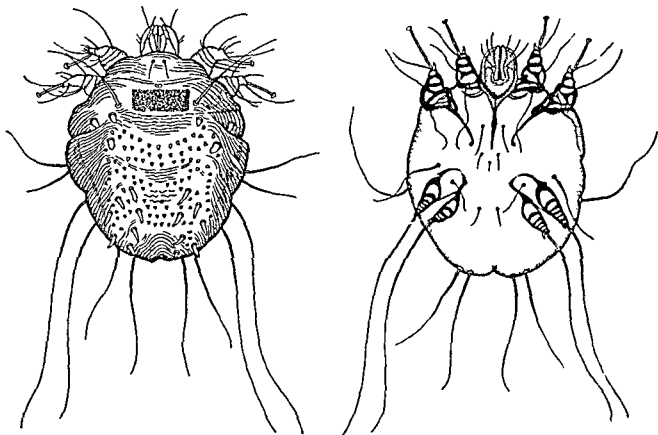
FIG 27 2 — Sarcoptes scabiei the sarcoptic mange mite Female Left, dorsal view right, ventral view (From Belding, 1952 Textbook of Clinical Parasitology Courtesy Appleton Century Crofts, Inc.)

After laying 10 to 50 eggs the female dies, she may usually be found at the end of a tortuous tunnel from 0 5 to 3 0 cm in length The eggs hatch in about 5 days and the larvae either remain in the parent tunnels, escape and wander back to the skin surface, or make new burrows The larvae transform into the nymphal stage, moult, and, finally, pubescent males and females are produced Mating occurs either in the moulting pockets or near the skin surface, whereupon the ovigerous females start out to make new burrows The complete life cycle from egg to ovigerous female may be completed in 10 days, though the average period is probably from 11 to 15 days

Although the mites do not reproduce except when on the host itself, they can live for 2 to 3 weeks when removed from hogs. Eggs or mites that become dislodged and drop in moist protected places may remain viable for 2 to 4 weeks in mild weather They are, however, very susceptible to desiccation and if in dry surroundings and

exposed to direct sunlight, it is unlikely that they will survive more than a day or so

Swine usually become infested with *Sarcoptes* by direct contact with infested animals However, infestation may occur when clean hogs are placed in pens and yards where infested swine have recently been kept The possibility that hogs may pick up the mite from contaminated premises should be kept in mind at all times

All types, breeds, and ages of swine are susceptible to sarcoptic mange, though well fed, healthy, and well cared for animals seem to have considerably more resistance to the devastating effect of the parasite than do unthrifty animals

The first lesions of sarcoptic mange are usually seen around the snout, eyes, ears, or any place where the skin is tender and the hair is thin In older pigs, lesions are frequently seen around the ears, tail, and on the inside of the hind legs, in the region of the groin and shoulder pits From
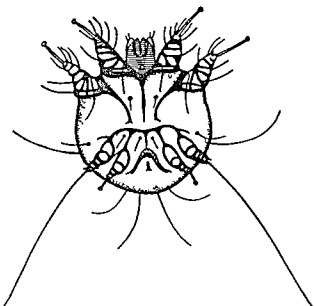
FIG 27 3 — Sarcoptes scabiei, the sarcoptic mange mite Male, ventral view (From Belding, 1952 Textbook of Clinical Parasitology Courtesy Appleton Century Crofts Inc)

these areas the mites spread and multiply until large areas of the skin become involved The period of development of lesions is extremely variable They may be observed 6 weeks after the original exposure, or a much longer period may elapse before their appearance

As a result of the sensitization of the infested animal, considerable irritation, itching, inflammation, and swelling of the tissues occurs The intense itching causes the animal to scratch and rub vigorously Scratching liberates the tissue fluids from the small vesicles around the burrow of the mite This serum coagulates, dries, and forms crusts on the surface of the skin Excessive keratinization and proliferation of connective tissue occurs, with the result that the skin becomes greatly thickened and wrinkled The thickened area becomes dirty and frequent rubbing of the hard scabs may give a glistening leathery appearance to the skin In advanced cases, the heavily scabbed areas may crack, blood and serum may ooze out, and an offensive odor may be noted from these moist lesions

One attack of the disease does not seem to confer any immunity since cured animals may readily become reinfested if exposed to mangy hogs

In these times, when the dermatitis of swine are being studied extensively, it is most important that a correct diagnosis of mange be made A positive diagnosis consists of the demonstration of the mange mites since they are the sole cause of the disease This is often difficult to accomplish, especially in the early stages of the disease or in long standing chronic cases where the skin is wrinkled into deep folds and is leathery in appearance

To demonstrate the mites, deep skin scrapings should be taken with a blunt edged scalpel The scraping should be sufficiently deep so that blood oozes from the traumatized area The scrapings are examined in sunshine or under artificial light by use of a low power magnifying lens A better method of examination is to transfer the scraping to a glass slide, add 1 or 2 drops of mineral oil or 10 per cent solution of sodium or potassium hydroxide, crush the scraped material with the flat side of the scalpel, spread the macerated material thinly, drop on a cover glass and systematically examine under the lower power of the compound microscope For greater concentration of mange mites, skin scrapings may be allowed to macerate overnight in 10 per cent solution of sodium or potassium hydroxide The material is concentrated by centrifugation, washed, and recentrifuged The sediment in the centrifuge tube is examined for the presence of mites

The demonstration of sarcoptic mites is not easily accomplished They are more likely to be found if scrapings are taken from areas around the margin of recent lesions where the mites may be actively at work and migrating to new areas. If ear lesions are present, the mites often may be more easily demonstrated from this area

Demodectic Mange

A minute mite known as *Demodex folliculorum* var *suis* or *Demodex phylloides* is the cause of demodectic mange in swine The species *Demodex folliculorum* includes varieties that are found on man, dog, sheep, goat, and other mammalian hosts.

The disease produced by this mite is referred to as follicular or demodectic mange Records of distribution of this parasite are few, as the symptoms of demodectic mange in swine are not marked A diagnosis of this condition is seldom made

These mites pass their lives in the hair follicles and sebaceous glands of the skin The closely related variety of *Demodex* in the dog has been found in the lymphatic glands and it has been suggested that this parasite spends part of its life cycle in the blood stream or internal organs

The demodectic mites are minute in size and usually measure about 0 25 mm in length (Fig 27 4) They have an elongated wormlike body divided into head, thorax, and abdomen the thorax bears 4 pairs of short stumpy legs The eggs are spindle shaped As with a great many of the mites the life cycle is not fully known
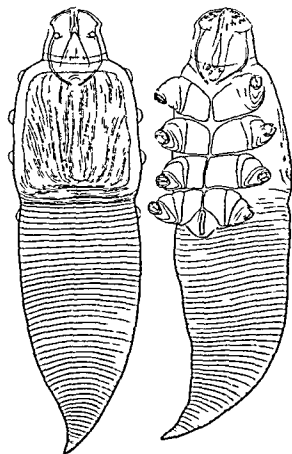
FIG 27 4 — Demodex phylloides, the demodectic mange mite Female Left, dorsal view; right, ventral view (From Hirst, 1922 British Museum, Economic Series No 13.)

When present in small numbers, these mites do not seem to cause the animal much inconvenience However, swine in a poor state of nutrition seem to allow an increase in the mite population which may give rise to well marked skin lesions The infestation usually is noted first in areas where the skin is soft and of fine texture such as around the snout and eyelids From these foci it spreads gradually to the underside of the neck, down to the abdomen and to the inside of the legs At first the skin becomes reddened and the affected areas become scurfy and scaly In later stages of the disease small hard nodules appear on the skin surface, ranging in size from a pin head to a pea As the condition progresses the nodules may rupture and yield a thick creamy pus or cheeselike material in which mites usually are found Two or more nodules may become confluent which upon rupture, leave small suppurating cavities

As in the case of sarcoptic mange a positive diagnosis is made by the demonstration of the demodectic mange mite This is accomplished by the examination procedure suggested previously for sarcoptic mange

Control of Hog Mange

Until recent years the control of sarcoptic mange in swine has been a problem The biology and habits of *Sarcoptes scabiei* make it difficult to combat these forms with an effective acaricide Sarcoptic mange is not easily eradicated To be effective treatments must be applied with extreme thoroughness Since current treatments for mange and lice are quite similar, the application of parasiticides for the control of mange will usually take care of the louse infestation

As listed by Cobbett (1956), the methods used commonly for the treatment of mange are dipping spraying medicated wallows, medicated bedding hog oilers and hand applications. The use of the last three mentioned may temporarily check the spread of mange but they are not wholly effective in its eradication

The older treatments consisted of the application of oily materials and lime sulfur solutions However, the newer series of chlorinated hydrocarbons such as benzene hexachloride (BHC), lindane, and chlordane in the form of wettable powders and oil emulsions are now widely used and have proved highly effective in controlling sarcoptic acariasis For detailed description of the use of oil preparations medicated wallows, construction of dipping tanks, and dipping procedures, the reader is referred to Kemper and Peterson (1955) For the control of mange using a chlorinated hydrocarbon dip or spray, the recom ... led concentration of gamma isomer of ... nzene hexachloride is 0 13 per cent ... uane is used at a strength of 0 5 per ... DDT is not recommended for the treatment of hog mange

It is generally considered that one thorough treatment using BHC, lindane, or chlordane will eradicate sarcoptic mange from swine However, in long standing chronic infestations when the skin is thickened and encrusted, a second treatment may be required A second application should be made about 10 days after the first treatment It is also important that all body surfaces including the skin inside the ears under the belly, and inside the thighs be completely covered with the parasiticidal mixture All animals in a herd *must* be treated

If the acaricide is applied as a spray, a few animals in a small enclosure should be treated at one time thus assuring a complete wetting of each individual Young hogs may be dipped manually in a tub or small barrel containing the acaricide

In order to permit a spray or dip to penetrate the mange lesions, it is desirable to keep treated animals out of the sun and wind for a few hours to permit slow drying

Because of the importance of demodectic mange in dogs most control procedures have been directed toward the alleviation and eradication of the disease in this animal It is generally considered that animals in a state of malnutrition are more sus ceptible to this disease A period of rest on a high nutritional level with adequate vitamin content should precede treatment

In swine no specific treatment for demodectic mange is known The use of dips or sprays with crude petroleum seems to check the spread of the parasite Probably the best recommendation for treatment is repeated applications of BHC or lindane as suggested for the control of sarcoptic mange Severely infested animals and those that do not respond to treatment should be removed from the herd and marketed The premises should be thoroughly cleaned and disinfected before healthy swine are admitted

FLIES THAT CAUSE MYIASIS IN SWINE

Myiasis is the term used to indicate the condition resulting from the invasion of tissues or organs of man or animals by dipterous larvae

Gasterophilus spp According to the host parasite check list of Dikmans (1945), gastric myiasis in swine due to the larval stages of *Gasterophilus intestinalis* and *Gasterophilus hemorrhoidalis* has been observed The finding of an equine parasite in this habitat should probably be considered as an incidental or abnormal parasitism

Cuterebra spp The larvae of the rodent botfly, usually found under the skin of such domestic animals as kittens and puppies has been reported by Dalmat (1943) from the throat and trachea of swine This also is suggestive of an accidental parasitism

Callitroga hominivorax This dipterous insect, commonly known as the screw worm, is an obligatory parasite of warm blooded animals that attacks swine The adult fly lays its eggs on all types of wounds but prefers fresh abrasions of any kind Upon the hatching of the eggs, a serious cutaneous myiasis is initiated During the winter months this insect is confined to the warm southern climate of the United States in parts of Texas Florida, Arizona, and southern California During the summer months the fly migrates northward and may even be found in the northern states

if the larval stages are transported north on infested animals

The adult fly is attracted by any fresh abrasion such as a wire cut, scratch, wound, tick or fly bite, and especially to the naval opening in the newborn The extent of the infestation, the tissues involved, and the depth of penetration of the dipterous larvae depend upon the number of active larvae and whether or not the infestation is treated

The adult screw worm fly is about twice the size of the ordinary house fly and bluish or bluish green in color The female oviposits masses of about 200 eggs on the edge of a fresh wound These hatch in 12 to 24 hours and the tiny maggots feed as a colony in the fresh tissues The larvae complete their growth in 5 to 10 days, drop to the ground and pupate The pupal stage lasts from 7 to 10 days but may last for several weeks if the weather is not warm enough for development The flies emerge, mate, and are ready to lay eggs when 5 or 6 days old If unfavorable weather conditions prevail, the time of development of the various stages of the life cycle may be greatly prolonged

A fly known as the secondary screw worm fly (Callitroga macellaria) is a blowfly which mimics the screw worm fly The larvae are found in swine in the same habitat as those of C hominivorax It is a secondary invader and selects decaying tissues and material upon which to lay its eggs The adults of both these flies are practically indistinguishable on casual inspection, the larvae may be readily differentiated by a specialist.

Treatment of screw worm infestations is accomplished by the proper and timely application of larvicides which are not toxic to the infested host An old remedy is the application of benzol to kill the larvae, but such a procedure does not protect the wound from reinfestation About 1940, a formulation known as Smear 62 was developed by the Bureau of Entomology and Plant Quarantine of the U.S.D.A and was found highly effective in controlling screw worm infestations This

medication will protect wounds for about 3 days, however, it is inferior to EQ 335 which was developed about 1950 The latter contains lindane, is less volatile than Smear 62 which contains benzol, and is more effective in that any screw worm flies that return to a treated wound will be killed through the residual action of the lindane Reapplication of both of these formulations may be necessary until wound healing has occurred

Prevention of screw worm infestations depends to a great extent on good management practices and the use of effective and approved insecticidal formulations Fences, corrals, and equipment should be kept in good repair so that hogs do not suffer cuts minor wounds, scratches and abrasions Cuts due to the milk teeth of suckling pigs and lacerations from rough handling may attract the adult screw worm flies Such procedures as castration and ear tagging should be performed during the cooler months of the year when the screw worm menace is at a minimum

If such recommendations cannot be fitted into the management program, all wounds should be treated with an approved screw worm remedy and animals temporarily placed in a hospital pasture where they can be examined daily and treated until wound healing occurs

FLEAS AFFECTING SWINE

Though lice are fairly host specific, fleas are not They are not permanent parasites and often attack animals other than their usual host. Unfed fleas may survive for several months off their host providing they are in a humid environment and have sufficient debris in which to hide

Both the human flea, Pulex irritans, and the stick tight flea, Echidnophaga gallinacea, are known to infest hogs The stick tight flea is a common pest of poultry in the southern United States but may become of great annoyance to man and hogs. The human flea is found on swine and breeds freely in the litter of hog houses. It also may become quite a serious pest.

Fleas are wingless insects with laterally

compressed bodies that measure about 2 0–4 0 mm in length The chitinous exoskeleton is usually a brown color Legs are long, strong, and well adapted for jumping

A blood meal seems to be essential before mating and egg production Under favorable conditions of humidity and temperature, and with frequent access to its host the adult flea may live for several months The eggs are pearly white in color, about 0 5 mm in length, and oval in shape They may be laid on the host but usually drop off or are laid in the host's nest or bedding The length of incubation s quite variable depending upon the cies and environmental temperature and humidity Upon hatching, a cream colored maggot like larva is liberated which feeds on dried blood from the feces of the adult flea and on organic debris On becoming full grown, a cocoon is formed within which the larva changes to the pupal stage, finally the adult flea emerges The entire life cycle from egg to adult could be completed in about 3 weeks under optimum conditions, though it is probable that the average time is considerably longer Barns and sheds which are used as sleeping quarters for hogs may become heavily infested and the occupants may suffer considerably from the irritating bites of this insect pest If control measures are not adopted, serious consequences may result especially in animals that are in ill health and unable to care for themselves

In attempting to control an infestation of fleas, it is important that attention be given to the breeding places as well as to the treatment of infested hogs All bedding, litter, trash, and dirt should be cleaned up and burned or treated so that the immature stages are destroyed

On the animal, control may be accomplished by the use of an insecticidal spray or powder Sprays made up with BHC or lindane should contain 0 06 per cent of the gamma isomer, DDT should be used at 0 75 per cent, and chlordane at 0 5 per cent For use as a dust, several insecticides are effective For many years derris

dust has been used effectively for the control of insects In the case of swine, a dust of derris or cube powder containing 1 per cent rotenone should be used Since fleas usually move actively over the animal, it is satisfactory to apply the dust only over the head and neck region and along the back Of the newer insecticides effective control may be obtained by using powders containing 10 per cent of DDT or methoxychlor or 1 per cent lindane or 5 per cent chlordane Again it should be stressed that fleas cannot be controlled effectively by treating only the host animal — the surroundings must also be cleaned up and an insecticide applied to kill the immature stages

For the control of infestations in hog pens sheds, and portable hog houses, litter should be swept up, removed, and burned Since DDT is highly effective in controlling the immature stages, a 5 per cent concentration should be sprayed or dusted over the area where the fleas have been propagating Chlordane at 2 per cent or dieldrin at 0 5 per cent may also be used on floors and in pens Infestations in yards, barns, or under hog houses may be controlled by dusting or spraying the area with wettable powders of DDT, lindane chlordane, or malathion Dusts are usually applied at the rate of 1 or 2 pounds to 1,000 square feet of surface, sprays are applied at the rate of 2 gallons of the formulation used to 1,000 square feet

MOSQUITOES AND FLIES ANNOYING TO SWINE

Mosquitoes are generally considered as pests of man but they also attack livestock causing discomfort, irritation, and, at times, serious losses In several parts of the United States, species of mosquito are quite important pests of hogs In Florida, species of *Aedes* have been observed attacking hogs in large numbers and it is probable that other species, especially the night feeders, are of considerable annoyance to swine

To control this pest, knowledge of the habits and breeding places of the larvae and adults is essential Several highly ef

fective insecticides are available for use in a control program

The stablefly *Stomoxys calcitrans* also known as the biting housefly or dogfly may cause considerable annoyance to swine during hot weather This fly is a vicious biter and may feed several times daily to obtain blood It has been suggested that this fly may also serve as a mechanical vector of certain infectious disease producing agents especially bacteria The preferred breeding places for this fly are wet straw manure and decaying vegetable matter Control measures should be directed toward good barnyard sanitation the elimination of breeding places and the use of insecticides on animals and in barns and hog sheds For use on hogs a synergized pyrethrum formulation as a water spray appears to give good results In sheds and barns DDT lindane or chlordane sprays may be used for effective control

TICKS AS PARASITES OF SWINE

As parasites of domestic and wild animals ticks are responsible for serious economic losses Not only do they cause great annoyance and irritation but they may serve also as vectors of many important rickettsial and protozoan diseases of man and animals Injury may result from their bites which may become secondarily infected from the inoculation of toxic substances or from severe blood loss if the ticks are present in sufficiently large numbers

Ticks are parasitic arthropods of the class ARACHNIDA and the order Acarina They are found on a wide range of host animals the majority of species do not appear to be very host specific Swine are not generally considered as being a usual host for these parasites though several different species have been reported from swine in North America

There are two large families of ticks the Ixodidae known as the hard ticks and the Argasidae or the soft ticks Several species of ixodid ticks have been reported as occurring on swine in this country They are

Dermacentor andersoni (Rocky Mountain Spotted Fever tick)
Dermacentor variabilis (American dog tick or wood tick)
Dermacentor nitens (tropical horse tick)
Amblyomma maculatum (Gulf Coast tick)
Ixodes ricinus scapularis (the black legged or shoulder tick)

Of the argasid or soft ticks only 2 species have been reported from swine

Ornithodoros turicata (relapsing fever tick)
Otobius megnini (spinose ear tick)

The body of the tick is usually oval or elliptical in shape and is covered with a tough leathery integument There is seldom any visible demarcation between the cephalothorax and the abdomen The attaching organ of the tick is known as the hypostome which is a club shaped structure armed with numerous rows of recurved teeth which permit secure anchorage for the tick The dorsal surface of the ixodid or hard tick bears a dorsal shield or scutum which is small in the female but covers almost the whole dorsal region of the male The scutum may be colored furrowed and highly ornate in some species There are 4 pairs of legs provided with various kinds of prolongations and spurs

There are 4 definite stages in the life cycle (1) the egg (2) the larva which is usually known as a seed tick and has only 3 pairs of legs, (3) the nymph and (4) the adult The larval tick on hatching from the egg attaches to a host and engorges with lymph and blood When fully engorged it moults and becomes a nymph which has the characteristic 4 pairs of legs The nymph then feeds on a host and when fully fed will moult and develop to a male or female adult Mating occurs in the adult female engorges and increases very considerably in size She finally drops off the host and finds a secluded spot to lay her eggs she then dies Since all ticks are dependent upon blood for their development anemia of the host will be seen if ticks are present in sufficiently large numbers. The tick saliva appears to be a local

irritant at the site of injection and may also be a systemic toxin. Most animals will tolerate a few ticks but, if the population becomes large, they will try to alleviate the irritation by rubbing, scratching, licking or biting. This usually results in injury to the skin surface, with secondary bacterial invasion and the development of lesions and raw areas.

Tick infestations may usually be seen upon gross visual examination of the host. They may be found on any part of the body surface but are often seen around the ears, neck, and flanks. Ticks may vary greatly in size since any stages of a tick developmental cycle may be found on the same animal.

Tick control is usually attempted by applying acaricidal substances directly to the skin of infested animals. If only a very few ticks are present on an individual, they may be removed manually. The new chlorinated hydrocarbon insecticides are now widely used. To a great extent, they have replaced such insecticides as rotenone, nicotine, and arsenic compounds which were in general use until about 1945. Tick control may also be accomplished by the application of acaricides to infested premises and to small areas as hedge rows, grassy plots, and small pastures. Acaricidal applications to large wooded or pasture areas are not practical.

The control of these pests on swine should be carried out on a herd basis. Formulations may be applied in the form of sprays or by dipping. Treatment aims to destroy the tick stages on the host animal and to leave residual insecticide on the animal to minimize reinfestation for a few weeks following the application.

According to McIntosh and McDuffie (1956), toxaphene is the formulation of choice for the control of ticks on livestock. Toxaphene is effective against all stages of ticks. It is available as an emulsifiable concentrate or wettable powder, both of which may be used in sprays or in dipping vats. Toxaphene is used at a concentration of 0.5 per cent and its residual action will aid in protection against reinfestation for 2 weeks or longer.

DDT is highly effective against ticks prior to feeding but is not effective against engorged ticks. However, since lindane is highly effective against engorged ticks but does not have the residual effect of DDT, the two are usually combined to give effective control. Sprays or dips containing 0.5 per cent of DDT and 0.025 per cent of lindane or the gamma isomer of BHC will give highly effective immediate kill with a good residual effectiveness. Both these insecticides are available as emulsifiable concentrates and wettable powders.

Chlordane is used also for tick control on animals at a concentration of 0.5 per cent. However, it is not used extensively due to the possibility of cumulative effects in the animal. The old time standard arsenical dip containing arsenious oxide is still used in the control of the cattle fever tick.

Sprays and dips are generally the most common methods of applying materials for tick control. Sprays are probably more widely used than dips, and if animals are sprayed thoroughly and carefully, effective control will result. Dips maintained at the proper strengths are highly effective, but in the treatment of swine a thorough spraying should give satisfactory control.

In the case of the spinose ear tick the nymphal stages are found most usually in the ears of swine. Hand treatment should be used for the control of this infestation. McIntosh and McDuffie (1956) recommend a formulation of 5 per cent of BHC (15 per cent gamma isomer), 10 per cent of xylene, and 85 per cent of pure pine oil. This mixture is applied directly to the inside of the ears by use of a squirting oil can or syringe to the tip of which is attached a small length of rubber tubing.

For the control of ticks in outdoor areas of small size, dust or sprays may be used with satisfactory results. A 10 per cent DDT dust is most commonly used for this purpose at the rate of 20-25 lbs. per acre

REFERENCES

COBBETT, N G 1956 Hog mange Yearbook of Agr U S D A p 347

———, AND BUSHLAND, R. C 1956 The hog louse Yearbook of Agr , U S D A p 345

DALMAT, H L 1943 A contribution to the knowledge of the rodent warble flies (Cuterebridae) Jour Parasit 29 311

DIKMANS G 1945 Check list of the internal and external animal parasites of domestic animals in North America Amer Jour Vet Res 6 211

FLORENCE, L 1921 The hog louse, *Haematopinus suis* Linné its biology, anatomy and histology Cornell Univ Agr Exp Sta Memoir 51

KEMPER, H E, AND PETERSON, H O 1955 Hog lice and hog mange U S D.A Farmer s Bull 1085

LAPAGE, G 1956 Monnig s Veterinary Helminthology and Entomology, 4th ed Williams and Wilkins Co, Baltimore

McINTOSH, A, AND McDUFFIE, W C 1956 Ticks that affect domestic animals and poultry Year book of Agr, U S D A p 157

SCHANG. P J 1952 La variole des porcelets Rep 14th Internat Vet Cong 2 121

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CHAPTER 28

Nematodes, Acanthocephalids, Trematodes, and Cestodes

NEMATODES

The roundworms of swine play a very definite role in the economy of the industry While often quoted figures of losses are mere "guesstimates," one has only to observe the necropsy of a few runt pigs with ascarid occlusion of the intestine to realize the loss that does occur Pigs 3 and 4 months old may be seen weighing 10 to 15 pounds When one considers the handling, feed, and the original cost of the animal, the total loss could mean the difference between success and failure to the swine raiser

Lungworms, threadworms, n o d u l a r worms, and also kidney worms take their toll in reduced weight gains and even death We have only begun to guess what effect the presence of these parasites may have on a host with a concurrent disease

Trichinella infections present a public health problem It has been estimated that 10 to 20 per cent of our population may be infected, however, this does not imply that number of clinical cases Nevertheless, sporadic epidemics occur with morbidity and even death to humans This can and should be prevented Public education concerning the proper consumption of pork products and the prevention of feeding raw garbage to swine has materially affected the incidence of trichinosis in recent years

At present, swine cannot easily be kept worm free, but the parasites can be con-

trolled to prevent serious loss of animal and human life

Ascaris lumbricoides var *suum*

Phylum NEMATHELMINTHES Bilaterally symmetrical, unsegmented round worms Complete alimentary tract with mouth and anus usually present Body cavity present but in most cases without lining membrane Both free living and parasitic forms

Family Ascaridae Head with three prominent lips supplied with papillae Mouth without chitinous buccal capsule, intestine simple and devoid of diverticula Male usually without caudal alae and rarely with precloacal sucker, two spicules usually equal or subequal, gubernaculum sometimes present Female usually ending conically, vulva usually anterior to middle of body, oviparous

HOSTS

Normal pig, abnormal apes, cattle, sheep, and squirrels There is evidence that the larval stages will undergo partial development and migration in almost any mammal host unfortunate enough to ingest infective eggs

There has been widespread acceptance of the idea that the ascarid of pigs is identical to that of man from a morphological stand point but different in its physiological requirements — so different, many feel that

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underwent a molt within the egg about
the 18th day Alicata (1934) then demon
strated that the egg was not infective until
after this molt

Once the first molt has occurred, the
eggs may be ingested and hatched in the
small intestine The larvae bore into the
mucosa enter the portal system, and may
enter the liver as early as 18 hours after
ingestion Within 5 or 6 days the larvae
have left the liver and have located in the
lungs In experimental infections in pigs
one often sees respiratory embarrassment
about one week after infection The lar
vae grow and molt for a second time while
in the lungs Near the tenth to twelfth
day a third molt occurs and most of the
larvae migrate to the trachea and are
swallowed Thus they reach the small in
testine where they make a fourth and final
molt and grow rapidly to adulthood In
initial field infections larvae are recovered
as early as 14 days from the small intestine

The usual time from egg stage to egg
stage is around 50 to 60 days

LESIONS AND CLINICAL SIGNS

The first noticeable symptom of *Ascaris
lumbricoides* infection in young pigs is a
soft, moist cough occurring about a week
after the pig is placed on an infective hog
lot or given an experimental infection
Studies in England (Betts, 1951) demon
strated a temperature rise to 105–106° F
starting around the fourth to sixth day
after heavy experimental infection and
lasting several days during the period of
coughing and then disappearing with the
cough He made the strong point that very
heavy doses of viable ascarid eggs did not
produce the pneumonia so often attributed
to ascarids The idea was presented that
persistent coughs in swine could be due to

cross infections are impossible Abdulrach man and Joe (1954) have demonstrated differences in the lip denticles of the human and pig form to support the idea of different species although admittedly there were intermediate forms in a few cases Whether such slight morphological differences will be accepted by morphol ogists as specific characters remains to be seen

Early workers (Ransom and Foster 1920, Koino 1922 Payne et al, 1925) established the theory that ascarids from man and pig were not interchangeable by means of in fection A German worker, Reiche (1921), did not agree and later De Boer (1935) came to the conclusion that cross infection from human ascarids to pigs can take place

The whole problem of cross infection needs more examination In recent years several workers have found great difficulty infecting pigs with their own ascarids With the advent of excellent isolation facilities and the raising of pigs taken from cesarian section for experimental purposes, the way has been opened to assure that animals are not harboring a migratory in fection prior to experiments Lindquist (1957a) noted that experimental infection of pigs with pig ascarids was not accom plished in spite of viability of eggs as tested through guinea pig passage Verbal reports of other investigators indicated the same difficulty If pigs are not easily infected with pig ascarids, is there any reason to believe that failure to infect pigs with human ascarids proves that human ascarids are physiologically different?

DISTRIBUTION

This parasite is found wherever swine are raised

ORGANS OR TISSUES INVOLVED

The small intestine is the normal location for adult worms, however, they are known to migrate into the common bile duct. The larval stages are transported via the blood or lymphatic systems and thus may be found in many tissues through out the body Larval stages generally, ac cording to their migratory pathway, in volve the liver and lungs most often It seems quite possible that larvae in the blood stream may be swept to remote organs where they may be discovered upon examination of tissue sections How often or how commonly this occurs is not known because of the lack of histological data on normal slaughters and the difficulty of identification of nematodes in tissue sec tion Quite probably, all of our domestic animals may be carrying "lost larval stages' of either their own normal helminths or those from some other host There is a need to study tissue sections for methods of identification of worms possessing migra tory phases All too often larvae in lung or liver tissue of swine are called ascarids merely because ascarid larvae are normally found in these locations Why couldn t they be hookworms of cattle? How many veterinarians, parasitologists, farmers, and others working closely with domestic animals may be carrying encysted larval forms of domestic animal helminths? Beaver (1954) convincingly showed the presence of dog ascarid larvae in various human organs as a public health problem Fortunately for both domestic animals and man, host specificity of many roundworms prevents the worm development, and the barriers of the skin, lungs, liver, and other organs are often capable of walling off the invader with little detriment to the host Kennedy (1954) showed evidence that swine ascarids were not only capable of migrating through the liver and lungs of cattle but were also responsible for a state of sensitivity with the development of gross visible lesions and production of an eosinophilia

MORPHOLOGY

Ascarids of swine are robust, cream colored roundworms, in their adult state measuring nearly a foot long in the case of the female and a thickness of about one-quarter inch The male is a few inches shorter and comparatively thinner The male posterior end is most frequently fish hook shaped, and sometimes its tiny brown copulatory spicules may be seen extended

from the posterior end of the worm Char acteristically, both sexes have three lips at the anterior end but these are not easily visible For a good fairly recent descrip tion, the reader is referred to Baylis (1936) Ascarids from cattle and horses superficially appear like those of swine When swine ascarids are encountered in sheep, they are usually much smaller and sexually un developed

The eggs of *Ascaris lumbricoides* are rather characteristic, thick shelled, brown ish, oval formed bodies measuring from 45 to 87μ in length by 37 to 57μ in width The size variation in the literature is con siderable The surface of the egg is covered with a bumpy irregular coating of sticky albuminous matter Normally the eggs examined from feces appear brownish in color although those taken directly from female worms lack this coloration Some attribute the brownish color to the bile encountered in the intestine It is not un usual to find swine feces containing eggs from infertile *Ascaris* females These eggs may present a bizarre, unpredictable shape They may be triangular, elongate, with or without albuminous coating, vacuolated, and otherwise different from the normal Otto (1932) presented an excellent photo graphic plate of these infertile forms Now and then, one encounters fertile eggs in the feces which are void of the albuminous coating

LIFE CYCLE

Although *Ascaris lumbricoides* was first described in detail by Linnaeus in 1758, much older writings mentioned this form or similar ones For many years it was thought that these ascarids developed di rectly in the intestine It remained for Stewart (1916) to show that they had a migratory phase He did this in rats and mice and, when the worms failed to de velop to maturity, concluded that the ro dents were an intermediate host Ransom and Foster (1917) and Ransom and Cram (1921) showed that the migration was normal in the pig

Eggs are passed in the single-celled stage Embryos develop in 9 to 13 days under

optimal conditions of temperature and humidity Oddly enough, Stoll (1933) found that embryonated eggs, in spite of appearing to have infective embryos failed to produce lung damage in guinea pigs until sometime after the 34th day of in cubation at room temperature Ransom and Foster (1920) indicated that at opti mum temperatures (30–33° C) the larvae underwent a molt within the egg about the 18th day Alicata (1934) then demon strated that the egg was not infective until after this molt

Once the first molt has occurred, the eggs may be ingested and hatched in the small intestine The larvae bore into the mucosa enter the portal system, and may enter the liver as early as 18 hours after ingestion Within 5 or 6 days the larvae have left the liver and have located in the lungs In experimental infections in pigs one often sees respiratory embarrassment about one week after infection The lar vae grow and molt for a second time while in the lungs Near the tenth to twelfth day a third molt occurs and most of the larvae migrate to the trachea and are swallowed Thus they reach the small in testine where they make a fourth and final molt and grow rapidly to adulthood In initial field infections larvae are recovered as early as 14 days from the small intestine

The usual time from egg stage to egg stage is around 50 to 60 days

LESIONS AND CLINICAL SIGNS

The first noticeable symptom of *Ascaris lumbricoides* infection in young pigs is a soft, moist cough occurring about a week after the pig is placed on an infective hog lot or given an experimental infection Studies in England (Betts, 1951) demon strated a temperature rise to 105–106° F starting around the fourth to sixth day after heavy experimental infection and lasting several days during the period of coughing and then disappearing with the cough He made the strong point that very heavy doses of viable ascarid eggs did not produce the pneumonia so often attributed to ascarids The idea was presented that persistent coughs in swine could be due to

FIG 28 1—Swine liver six weeks after natural infection of Ascaris lumbricoides was initiated

virus pneumonia or other causes rather than ascarid infection This phenomenon, short transitory coughing period, has n observed in experimental infections in the author's laboratory The above views are not in total harmony with descriptions of the symptomatology described in many texts but should be given careful consideration There is a need for further study in the laboratory and with field pigs subjected to low levels of infection over a longer period of time It is a unique and interesting finding that doses as high as 500 000 viable eggs produced no pneumonia and only a transitory cough

Failure to gain, lack of appetite, unthrifty appearance, an occasional icteric pig all may be symptoms of ascariasis The presence of runts in litters may also be indicative of this disease

Actual lesions initially appear in the liver (Fig 28 1) and the lungs The liver shows scarified mottling as early as two weeks after infection If lesions are numerous at slaughter, the entire organ may be condemned Information is lacking about these lesions For instance, the relationship of the number of lesions to the infective dose has not been ascertained Do these scars remain stable throughout the life of the pig, or is there some regeneration of liver tissue obliterating them in older swine? How does one explain the presence of many mature ascarids at necropsy and no liver scars at all? Recently a 10 pound runt pig necropsied in this laboratory contained 264 nearly adult ascarids with no liver scars visible (Fig 28 2) Possibly the larvae all entered the lymphatics and bypassed the liver, or perhaps the migratory phase of the infection was spent in another host later consumed by the pig At any rate, experimental answers are needed to these questions One thing that has always perplexed the writer is the absence of detailed descriptions or illustrations of liver scars in human ascariasis although the standard texts tell of liver migration and a life cycle identical to the swine form

The lungs of swine show petechial hemorrhages a few days after infection Here the amount of damage and produc

FIG   28 2—Swine **liver** showing absence of liver scars with the presence of 264 ascarids (at left) in the host

tion of pneumonia is open to further investigation What part the lung migration may have in exciting or fulminating low grade virus pneumonias needs to be investigated

There is very little damage in the intestine except when numbers of worms occlude the lumen completely (Fig 28 3) or occasionally mechanically perforate the gut

Spindler (1948) found that adult worms did migrate, at times plugging the bile ducts and causing a generalized jaundice or icterus with the resultant condemnation of the entire carcass In a large number of carcasses condemned he found about 8 per cent were due to this phenomenon

DIAGNOSIS

Since many swine pass ascarid eggs the presence of eggs alone is not diagnostic of clinical disease One must consider the total symptomatology The presence of adult ascarids scattered on the pasture or in pens is a warning of danger Swine often may shed spontaneously part, if not all, of their worm load as they approach maturity, however, an ascarid condition in the herd may mean trouble for ensuing litters On the other hand, young pigs may be loaded with worms in the migratory phase, be clinically ill, have a raised temperature, be off feed, and yet have negative fecal

FIG 28 3—Total occlusion of lumen of the intestine due to ascarids.

examinations Female ascarids are prolific egg producers said to lay over 200 000 eggs per day There is no level of eggs per gram of feces known to constitute a clinical level since many variables enter into this kind of determination The presence of large numbers of eggs, with or without clinical symptoms requires immediate attention To wait for symptoms is to wait for disaster since loss in feed and weight gain is costly to the producer Good husbandry in today's competitive market demands a routine worming program

TREATMENT

Although many preparations have been used at one time or another only those commonly used today will be discussed

1 Oil of Chenopodium Known also as wormseed is said to have been used by the American Indians in the days of Columbus It is an essential oil obtained from Chenopodium anthelminticum The plant is an annual or perennial herb originally from South America but it now grows in the United States Its most active ingredient is a fraction known as ascaridol From in vitro studies, its mode of action appears to be a penetration of the cuticle of the worm resulting in stimulation first and then a paralysis of the musculature with the result that the worm cannot resist the peristaltic movements of the intestine

Administration Animals are fasted 18 to 24 hours prior to administration of 2 to 4 cc per 100 pounds of body weight and followed immediately with about 60 cc of castor oil It may be given by dose syringe stomach tube capsule or even with milk or thin slop for herd use Some of today's preparations are put up with the drug right in the castor oil but Epsom salts are sometimes recommended following treatment The purge serves two purposes, one being to help remove the worms in their state of relaxation and the other to remove any remaining drug thus reducing toxicity The worms removed are not killed or otherwise damaged The eggs of the expelled female worms are still viable and capable of producing further infections

dvantages Very few today It is a safe, reliable anthelmintic.

Disadvantages It is most effective when given as individual doses and even then does not compare in efficacy with the newer anthelmintics It requires more work on the part of the veterinarian It has been shown to have an efficacy of about 74 per cent, which is considerably lower than some of the following drugs

2 *Sodium Fluoride* The need for an anthelmintic that could be successfully administered in the feed prompted Haber mann *et al* (1915) to demonstrate the uses of this chemical Their findings indicated that a 1 per cent amount in the feed for 1 day would produce an efficacy of 97 per cent This was based on a group of 52 pigs Later the same year this efficiency was substantiated by Enzie *et al* (1915) giving a 98 per cent figure and using 124 pigs Allen (1915) and Allen and Jones (1916) reported using 1 per cent sodium fluoride

Administration Animals should be put on dry milled feed for a day or so before this anthelmintic is used A 1 per cent sodium fluoride mixture is fed in dry feed for 1 day It is important to have adequate feeders to minimize crowding If possible, pigs should be separated according to size and about one feeding space allotted to each 1 pigs if self feeders are used A good general plan of worming is to worm pigs shortly after weaning and again about a month later if they are known to be subjected to worm burdens Sows and gilts can be wormed just before breeding There is no experimental evidence on the best time to worm sows some texts indicate it should be no later than the first half of pregnancy

dvantages Sodium fluoride today is surpassed by no other anthelmintic in effective removal of ascarids It is also the least expensive, it present, to use No purge is required

Disadvantages Sodium fluoride is more acutely toxic than other preparations on the market today, and it is also less palatable Feed must be totally dry so that the chemical can be thoroughly mixed, failure

to mix thoroughly may result in fatal poisoning Turk and Hale (1956) found that swine went off feed during treatment and showed weight losses Kelley *et al* (1956) using cadmium compounds, sodium fluoride, and piperazines indicated that at the age of 154 days the weights of pigs treated with these compounds were not significantly different from those of untreated controls or of those fed skim milk This lack of difference may, of course, be a function of the initial worm burden of all the animals in the experiment

3 *Cadmium oxide* This compound was reported as a successful ascarid anthelmintic in 1955 by Burch and Blair

Administration It is used at the level of 0 015 per cent in dry feed for a period of 72 hours Worms are expelled for a period of a week to 10 days after treatment Enzie and Colglazier (1955) reported a 91 per cent efficacy or better with this compound Advantages and disadvantages of cadmium compounds will be discussed in general after the next one is introduced

4 *Cadmium anthranilate* Guthrie (1951a, b) observed the efficacy of this anthelmintic to be, on an average, 93 9 per cent

Administration Cadmium anthranilate is generally used at the level of 044 per cent in wet or dry ground feed for a period of 3 days Elimination of ascarids continues for 10 days or longer after treatment

Advantages of cadmium compounds Cadmium is more palatable and not so acutely toxic as sodium fluoride It can be used in wet feeds and does not interfere with weight gains No purge is necessary

Disadvantages of cadmium compounds Cadmium is more expensive, at present, than sodium fluoride It must be used over a 3-day period Since cadmium tends to build up in the tissues, it is generally recommended for only one treatment during the life of the animal Cadmium is slowly dislodged from tissues, so animals should not be marketed for about 30 days following treatment Cadmium in the tissues could create a public health problem

5 *Piperazines* Since this simple ring compound is rather easily substituted, it

is impossible to discuss individually the growing number of different substitutions that are being reported. At present, there are several on the market for use with swine: piperazine citrate, adipate, 1-piperazine carbodithiotic, and recently Guthrie (1956) reported successful use of the sulfate and hexahydrate with swine.

The mode of action is said to be that of a depressant on the ascarids causing them to be flushed out while in a paralyzed state.

The effectiveness of certain of these compounds tested in swine is equal to that of sodium fluoride or the cadmium preparations. Apparently, this group is the least toxic of the swine anthelmintics and is not contained long as such in the tissues. Some workers have indicated activity of these compounds against nodular worms but such observations have been on a limited basis with little critical data on effectiveness.

Administration: This depends on the compound used. Several are soluble in water and may be used that way. Some are used in varying concentrations in the feed.

Advantages: Piperazine compounds, fed wet or dry, have little or no toxicity to swine. They are very palatable with a high per cent efficacy. They can be given on a 1-day schedule and repeated if necessary. They are effective in other farm hosts.

Disadvantages: At present, the cost of piperazine compounds is considerably higher than that of other anthelmintics although this may become lower with volume sales.

There is some optimism that certain piperazine formulations may be used prophylactically at low levels in the feed. In preliminary experiments, Lindquist (1957b) found that it was possible to feed one-tenth to one-fifth therapeutic dose per day over a period of 6 weeks without altering weight gain or general health. When these pigs were exposed to infective pastures, the drug did not prevent the migratory phase of the life cycle. However, it may be found that animals treated longer will lose the intestinal phase of the worms

before they develop to maturity. Potentially, this could reduce pasture or lot contamination. Data must yet be collected to establish that fact.

6. *Antibiotics:* At the time of this writing, a new antibiotic, hygromycin, has been developed and promoted as an efficient treatment for ascarids as well as other intestinal parasites. It is said to have low toxicity and is recommended as a low level additive to the feed.

PREVENTION

The McLean County system of swine sanitation has in principle the proper elements to control ascariasis, but certain practices have embellished its effectiveness. The system has four major points:

1. Farrowing pens or houses should be thoroughly scrubbed before farrowing time. Lye and very hot water are most often used at the concentration of 1 lb. of lye to 30 gallons of very hot water.
2. The sow should be scrubbed with soap and water thoroughly before being placed in the dry pen or house.
3. The sow and pigs should be hauled to a "clean" pasture at the appropriate time.
4. Old hog lots or permanent pastures should not be used.

One of the practices that has developed along with the program is the worming of the sow prior to scrubbing and placing in the farrowing pen.

The matter of "clean" or "worm-free" pastures is the most difficult part of the program and cannot be well observed. Spindler (1940) showed that ascarid eggs remained viable on Maryland pastures for 4 years (at which time the experiments were discontinued) with plowing twice a year and grain cultivation. Lindquist (unpublished data) has found that ascarid eggs on unplowed lots in Michigan have remained viable and infective for over 6 years. At the present time there is no method suitable to detect or measure the degree of infectivity of swine pastures.

It should be borne in mind that promiscuous use of pastures for worming swine is at the same time seeding that pasture

with hundreds of thousands of worm eggs None of our commonly used anthelmintics kills the eggs of worms shed from swine These eggs are disseminated on the lot or pasture adding to its infectivity Even if the pigs eat the shed worms, the eggs may remain viable and pass back to the pasture Worming programs might well be carried out in lots not to be used for pasturing young pigs

Metastrongylus elongatus — Lungworms of Swine
M pudendotectus
M salmi

Family Mestrongylidae Body usually filiform Mouth with or without very feeble buccal capsule Bursa somewhat reduced with more or less typical rays Parasites of the respiratory and circulatory system of mammals

HOSTS

The pig is normally the host for *Metastrongylus*, but Chandler (1955) mentions that *M elongatus* has been reported three times from man Schwartz and Alicata (1934) reported infections to maturity (not egg laying maturity) in the guinea pig and dog

DISTRIBUTION

Monnig (1947) indicates a cosmopolitan distribution Incidence figures may depend to a great extent on husbandry practices In England, Dunn et al (1955) found 20 5 per cent of 1,722 pigs infected Previous to that time Sullivan and Shaw (1953) in Oregon found 51 9 per cent of 518 market weight pigs infected Andrews (1956) stated that investigators in the southeastern United States found 70 per cent of swine examined had lungworms

MORPHOLOGY

These are long, slim nematodes acting and looking much alike *M salmi* has been found only a few times in the United States A description of the genus will be given followed by a differential table (Table 28 1) for the species

Metastrongylus Mouth with two lateral trilobed lips Bursa small All bursal rays very stout except the dorsal and externodorsal Tip of the lateroventral ray curves away from the ventroventral ray There is a large lobulated end on the anterolateral ray Posterolateral ray represented by a small branch arising from the mediolateral ray Spicules are long and slender with striated alae ending in a single or anchor like hook Posterior end of the female recurved ventrally Vulva in front of the anus Uterine branches parallel Eggs containing embryos when laid

Of the three species it may be said that they are almost impossible to differentiate Two of them, *M salmi* and *M elongatus*, have overlapping measurements while *M pudendotectus* is slightly larger They are uniformly embryonated when laid, and the shell is usually thick with slight mamillations Schwartz and Alicata (1934) have reported 'thin shelled eggs which were thought to represent very early eggs in which a condensation of shell material had not yet occurred

The longevity of eggs has been remarked upon by the Hobmaiers (1929), who observed eggs to have viable larvae after 3 months in a moist medium In the earth worm, the intermediate host, Spindler (1938) found larvae viable after 4 years in Maryland pastures

LIFE CYCLE

The adult worms live in the bronchi and bronchioles of the lung, a favorite locale according to Dunn et al (1955) being the caudal end of the diaphragmatic lobe (Fig 28 4) The adult worms apparently orient themselves head down or facing the terminal branches of the trachea where, according to Soliman (1951), they ingest inflammatory exudate as it is coughed up Embryonated eggs are either coughed or ciliated up, are swallowed, and are finally passed in the feces Any one of several species of earthworms is necessary for further development of the lungworm Upon ingestion of the embryonated egg by the earthworm, the first stage larvae hatch

TABLE 28 1

DIFFERENTIATION OF THE SPECIES OF *Metastrongylus**

		Metastrongylus elongatus	*Metastrongylus salmi*	*Metastrongylus pudendotectus*
Males	Length of spicules	3 9 5 5 mm	2 1 2 4 mm	1 4–1 7 mm
	End of spicules	Hook like	Hook like	Anchor like
	Gubernaculum	Absent	Absent	Present
	Genital cone	Strong	Moderate	Weak
Females	Length of vagina	Over 2 mm	1 2 mm	Less than 1 mm
	Provagina	Absent	Absent	Present
	Prevulvar swelling	Often set off sharply from body anteriorly projecting posteriorly and ventrally	Only slightly or not at all set off from body anteriorly projecting posteriorly	Set off sharply from body anteriorly with provagina attached
	Position of vulva	At posterior end of prevulvar swelling usually at juncture of swelling with body	Midway between anterior and posterior ends of prevulvar swelling pressed against ventral side of body	At posterior end of prevulvar swelling surrounded by provagina

* Modified slightly from Dougherty (1944)

from the egg and penetrate the posterior esophagus of the earthworm Some larvae enter the blood system and localize in the hearts In these positions the larvae molt twice during growth to become infective third stage larvae in something over two weeks Temperature and other environmental factors influence this maturation Pigs swallowing the earthworms quickly

digest free the infective larvae which penetrate the intestinal wall apparently following the course of the lymph For a time after infection larvae are found in the lymph glands of the pig Many escape from this location enter the blood stream and proceed to the right side of the heart and lungs where they break out to the air passages Apparently during this migratory

FIG 28 4—Lungworms congregated in the posterior tip of the lung

phase before reaching the lungs, the larvae complete two more molts making them young adults about the time they enter the lungs It is said (Schwartz and Alicata, 1934) that embryonated eggs may be present in the feces of the pig as early as 24 days after infection

The life cycles of the three lungworms so closely parallel each other that they may be discussed together here

LESIONS AND CLINICAL SIGNS

The actual necropsy lesions are often inconspicuous There may be some wedge shaped areas of vesicular emphysema along the ventral border of the diaphragmatic lobe near the caudal extremity The bronchi are thickened and dilated There are sometimes firm grayish nodules near the emphysematous areas There may be a hypertrophy of the bronchiolar muscle with a hyperplasia of the peribronchiolar lymphoid tissue

There is little or no damage to the enteron by penetrating larvae although there is probably a stimulation of lymphoid nodules along the intestine and mesenteries

Dunn (1956) has indicated occasional milk spots' on the liver perhaps due to accidental migration of larvae through the liver

The presence of eosinophils in the lung lesions may be pronounced Lymphoid development in the lungs appears to be present only in cases of long standing infection

In younger pigs, a parasitic pneumonia may be present and giant cell formations associated with the presence of large numbers of lungworm eggs liberated into the alveoli

The association of pneumonia and lung worm infections needs further study Particularly is this true in view of the fact that lungworms were demonstrated by Shope (1911) to be a reservoir for swine influenza virus This remarkable finding helped to explain the occasional outbreaks of interepizootic influenza in swine Perhaps lungworms act in some similar fashion in the case of virus pneumonia

Symptoms of lungworm disease in themselves may not be pathognomonic Severe coughing, difficult breathing, loss of appetite may all be evidence of lungworm disease Sullivan and Shaw (1953) did show that severe infection can be lethal On the other hand, they pointed out that subclinical infections failed to show significant differences between infected and control animals in respect to market time and carcass

DIAGNOSIS

The presence of lungworms can, of course, be ascertained by fecal examination but not consistently In the literature there is a paucity of actual egg counts that are linked with clinical disease This may be due to sporadic expulsion of eggs to the intestinal tract of the pig The presence of numbers of eggs with vaguely characteristic symptoms must serve as the basis for diagnosis

Dunn et al (1955) have indicated the use of a saturated magnesium sulfate solution (sp gr 1 285) to be superior to sodium chloride as a levitation fluid The method used was to shake a 2 gram sample of feces with 30 ml of the magnesium sulfate solution, sieve through a screen of 44 mesh per inch into two 15 ml centrifuge tubes, and centrifuge with coverslip for 8 minutes at 1,500 r p m The covers were then counted, and for more accuracy the tubes were trimmed with additional solution and second coverslips added with another centrifugation and count.

TREATMENT

At the present time, no commercial compound is available that is capable of removing or killing lungworms successfully Most efforts have been aimed at prevention because it was believed that any drug capable of destroying the worm in the lung would probably destroy lung tissue too [1]

PREVENTION

Control programs center around preventing contact between young pigs and earth

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[1] However, a compound cyanacethydrazide, tested in Britain is said to cause lungworms to move anteriorly where they can be coughed up At present its use is in the experimental stage.

LIFE CYCLE

The life cycle of nodular worms in swine has been established over a period of years by Goodey (1924, 1926), Alicata (1933), Spindler (1933), and Shorb (1948).

The eggs passed in the segmented stage hatch in 24 to 48 hours. Two molts take place in 3 to 6 days providing third stage infective larvae. When ingested by the pig, the larvae proceed to the large intestine. There is no evidence that the larvae can use any other route but oral for entrance to the host. It is doubtful if the infective larvae can withstand severe wintering but Morgan and Hawkins (1949) state that under optimum culture conditions they may live as long as 10 months. As early as 20 hours after ingestion larvae are found encysted in the mucosa and submucosa of the large intestine. Shorb (1948) indicated that within 6 to 10 days after infection, fourth stage larvae emerged from the nodules to occupy the lumen of the large intestine. The implication may be made that a third molt occurred some time within the first 10 days. The transition from the fourth to the fifth, or adult, stage by the final molt has not been traced. Sexual maturity in the case of *O quadrispinulatum* is known to take place 50 to 53 days after infection.

LESIONS AND CLINICAL SIGNS

Undoubtedly, few if any veterinarians or pathologists have seen pure infections of these worms producing clinical cases, so one must record the lesions and symptoms as they have been produced experimentally.

The lesions and their development are open to much more study. Goodey (1926) indicated from experimental infections the absence of nodules such as occur in sheep. Later Schwartz (1931) pointed out that he found nodules in field cases. It would appear that lesion development occurs similarly to that in sheep, original infections producing little or no reaction and secondary or tertiary infections stimulating greater production of nodules. Spindler (1933) pointed out that nodules may practically disappear after emergence of the larvae.

All available evidence indicates that these nodular worms do not produce in swine the extensive damage they do in sheep. The nodules in swine seem confined to the large intestine, seldom, if ever, totally disrupting the function of that organ. There has been discussion concerning the production of small ulcers and the role played by secondary bacterial infection, but little experimental information is available to support or deny such actions.

It has been said (Shorb, 1948) that there is hypertrophy of the regional lymph nodes, thickening of the intestinal wall, and production of a diphtheritic membrane, as well as edema of the mesocolon. The nodules themselves seem to be made up of a smooth homogeneous substance containing neither nuclei nor striations and to be surrounded to some extent by fibroblasts. No other organs of the body have been reported involved.

The symptoms have been described as anorexia, constipation, sometimes diarrhea and emaciation. Resultant death has been produced experimentally.

DIAGNOSIS

It is doubtful if diagnoses prior to necropsy are made often. The eggs may be confused easily with at least two other nematode parasites of swine. Identification of cultured larvae has been suggested as a means of diagnosis, but this is seldom used and is not practical as a field adjunct. The symptomatology is not pathognomonic. Necropsy examination appears to be the only certain way to determine the disease.

TREATMENT

Phenothiazine is, at present, most commonly used at the rate of 0.1 gm per pound of body weight orally to a maximum dosage of 20 gm. This may be mixed with ground feed or given individually in gelatin capsules. Such treatment has been reported to have better than 90 per cent efficacy.

Phenothiazine can be toxic to swine, the drug is known to produce paralysis and other toxic symptoms, especially in very young or weakened animals.

A word must be mentioned in passing about the use of piperazines for *Oesophagostomum* sp Although several preparations are labeled for use against nodular worms, there has not been sufficient published evidence to appraise critically its effectiveness in all the various forms Leiper (1954) found 86 5 per cent of the nodular worms were removed by use of polymeric piperazine 1 carbodithioic acid If all the piperazines prove to be that efficacious, a definite advantage occurs in that both *Ascaris* and *Oesophagostomum* may be treated in one operation

PREVENTION

The general pattern of swine sanitation is a fairly effective preventative Since the infective larvae live for months under optimum conditions, these worms are a more serious problem in the warmer climates Alicata (1955) found that 20 days after treatment of soil with sodium borate at the rate of 5 lb per 100 sq ft, the viable larvae in test baskets had been reduced to a very small number compared to the controls This may prove to be an available preventative When it is used for the control of kidney worm, it also provides nodular worm reduction

Hyostrongylus rubidus — Red Stomach Worm of Swine

Family Trichostrongylidae Small slender worms Mouth without cutting organs or leaf crown Buccal capsule vestigial or absent in adults Bursa of male well developed with large lateral lobes but an insignificant dorsal lobe

HOSTS

Swine are the only known host for *H rubidus*

DISTRIBUTION

The species is widespread in the United States Incidence reports that have been made in this country came from the South primarily, although the nematode has been seen in the northern states The author has collected it several times from swine in Michigan, but the origin of the animals was not known Other countries reporting its presence are England, Germany, Australia Hungary, and in 1940 Porter indicated its presence in Asia and Central America

ORGANS OR TISSUES INVOLVED

The stomach appears to be the only organ involved

MORPHOLOGY

Small slender red worms, the male measuring 4–7 mm long and the female 5–9 mm in length Adult worms possess a cephalic button formed by the cuticle and limited posteriorly by a definite groove There are backward directed cervical papillae about 4 mm from the anterior end

The male has a well developed bursa the two lateral lobes being continuous anteriorly Spicules are equal short and tapering to a point with a wavy ridge running the length and supporting a curved membranous portion which terminates in a second point A long narrow gubernaculum is present as well as a ventral arched structure known as a telamon The spicules measure 0 13 mm in length

The female has the anus 0 68 mm from the tip of the tail and the vulva 1 3–1 5 mm in front of the anus Just caudal of the vulva is a semilunar fold of the cuticle The vagina is bottle shaped and at right angles to the cuticular wall

The eggs are of the typical strongyle type, however, White (1955) pointed out an error of measurement in the original description (Hassall and Stiles 1892) White found the eggs to measure 70–76μ in length by 36–39μ in width He further pointed out that this means that differentiation from *Oesophagostomum* eggs is very difficult Fecal culture of larvae offers a means of differentiation but is not practical for field examinations

LIFE CYCLE

Adults inhabit the stomach of swine Eggs are passed in the feces unembryonated appearing 20 to 25 days after infection Experimentally eggs hatch 39 hours after being passed There appears to be no recorded evidence of parasitic de   it in the pig, although Alicata

(1935) found they could develop to maturity in the guinea pig in about 19 days, although no mention of "egg laying maturity in this host was noticed

LESIONS AND CLINICAL SIGNS

Several investigators have remarked that lesions varying from slight hyperemia to eroded areas or ulcers are produced by *Hyostrongylus* and there is general agreement on this fact Apparently, these are the only lesions noted Differences of opinion appear on the pathogenic significance of these worms Swine mortality has been attributed to this worm On the other hand Porter (1940) found no clinical evidence of injury nor weight loss resulting from infections Symptoms which have been recorded are wasting, incoordination, The serious clinical cases recorded may have been related to lactation periods nutritional deficiencies, or other disease conditions

DIAGNOSIS

It is doubtful if a diagnosis of stomach worm disease can be made other than by necropsy Determination of the presence of stomach worms can be made by culture of eggs in feces and examination of resultant larvae

TREATMENT

One of the only critical studies done on this species was that of Bozicevich and Wright (1935) They recommended administration of carbon disulphide either by stomach tube or capsule to animals fasted 30 to 48 hours prior to treatment The suggested dose was 0 1 cc per kilogram of body weight or 4–5 cc per 100 pounds of body weight This dose in their hands proved to be about 86 per cent efficacious

PREVENTION

Porter (1939) indicated that strict adherence to the sanitation system produced pigs with lower incidence and lower per centage of heavy infections Alicata (1935) experimentally showed that infective larvae could neither withstand −20° C for 9 hours nor drying for 240 minutes From these data one might suspect that *Hyo*-

strongylus larvae cannot withstand over wintering in the northern states

Ascarops strongylina— Physocephalus sexalatus— Thick Stomach Worms

Family Spiruridae Mouth usually with tri lobed lateral lips Sometimes lips are absent or small ventral or dorsal lips present A chitinized cylindrical vestibule usually found behind the mouth Esophagus long, divided into an anterior muscular and posterior glandular portion Cervical papillae present *Male* caudal alae well developed and supported by pedunculated papillae of which there are usually 4 preanal pairs *Female* vulva near middle of the body, oviparous

HOSTS

The adult is found in the stomach of swine *Physocephalus* has the ability to encyst in the stomach of a number of hosts and remain dormant until eaten by swine

DISTRIBUTION

Both of these worms have a very wide distribution, appearing in Africa, Asia Australia, Europe, and North and South America Incidence figures seldom have been given Spindler (1942) indicated that the incidence of these worms in swine in certain midwestern states has been found to be as high as 90 per cent of the animals examined In the same report a 50 to 80 per cent incidence was recorded in the southeastern states On the other hand, the writer has never encountered these two worms in necropsy examinations of pigs in Michigan

ORGANS AND TISSUES INVOLVED

The mucosa of the stomach appears to be the only tissue and organ involved

MORPHOLOGY

Ascarops strongylina Small red-colored worms A narrow cuticular wing is found on the left side running from a point about 3 mm from the anterior end and ending about 2 mm from the posterior end Below the lips and projecting into the buccal

cavity are two chitinous teeth formed by a prolongation of the wall of the pharynx The pharynx is marked by a series of chitinous ridges forming a continuous spiral

Male is 10–15 mm long possessing bursal wings or alae These wings extend to the posterior tip with the right one about twice as wide as the left Five pairs of somewhat asymmetrical stalked papillae support the bursa Spicules unequal the left being 2 24–2 95 mm long and the right 457–619μ long

Female is 16–22 mm long Vulva slightly anterior to the middle of the body Anus 215–275μ from the caudal tip

Eggs oval, 34–39μ long by 20μ wide with thick shells Embryos well developed in the shell before oviposition

Physocephalus sexalatus Small red colored worms Head with two tri lobed lips Head is marked off from body by a cuticular inflation ending in a circular de marking margin just anterior to the posterior end of the pharynx The pharynx contains a spiral band of 21 to 25 turns sometimes broken into discrete bands There are three lateral cuticular wings starting at the base of the cephalic inflation and extending about one third of the body length

Male is 6–9 mm long Narrow bursal membranes are present which are supported by 4 pairs of long stalked preanal papillae Postanal papillae (4 pairs) are near the tip of the tail and short The entire male tail is twisted about three turns Spicules unequal, the longest being 2 1–2 25 mm , the shorter one 300–350μ long

Female 13–19 mm long Anus 120μ from the caudal end Vulva is posterior to the middle of the body

The eggs are 22–26μ wide by 41–45μ long and slightly flattened at the poles Embryos are well developed in the shell prior to oviposition

LIFE CYCLE

The life cycles of these two worms are apparently quite similar Various species of dung beetles consume the eggs The larvae hatch and develop in the body cav

ity within a cyst to the third infective stage According to Alicata (1935) this may take 28 days or longer in the case of *Ascarops* and 36 days or longer for *Physocephalus* It is known that both species of stomach worms are capable of utilizing transport hosts but little is known of their actual parasitic development in the stomach of swine

LESIONS AND CLINICAL SIGNS

Foster (1912) indicated from the work of others that a pseudomembrane may be formed at the pyloric end of the stomach under which these worms are found partly attached to the stomach wall Red patches the size of a penny appeared around the pin prick opening made by the worm It has been suggested that the membrane is formed when the worms inoculate the mucosa with *Spherophorus necrophorus* bacilli which are commonly present in the stomach It has also been indicated that the worms may produce a gastritis and small ulcerations There are probably no pathognomonic symptoms present to aid in diagnosis

DIAGNOSIS

Characteristic eggs in the feces and necropsy examination appear to be the only way to tell if this infection is present

TREATMENT

There is very little experience on record due to the failure to diagnose cases while alive, however, Bozicevich and Wright (1935) found that carbon disulphide given by capsule or stomach tube was effective against *Ascarops strongylina* when used after fasting 36 to 44 hours at the level of 0 1 cc per kilogram of body weight No data have been recorded for *Physocephalus*

PREVENTION

Porter (1939) found that close adherence to the swine sanitation system markedly reduced incidence of all three swine stomach worms

Stephanurus dentatus — Kidney Worm of Swine

Family Strongylidae Is given under *Oesophagostomum*

(1935) found they could develop to maturity in the guinea pig in about 19 days, although no mention of egg laying maturity in this host was noticed

LESIONS AND CLINICAL SIGNS

Several investigators have remarked that lesions varying from slight hyperemia to eroded areas or ulcers are produced by *Hyostrongylus* and there is general agreement on this fact. Apparently these are the only lesions noted. Differences of opinion appear on the pathogenic significance of these worms. Swine mortality has been attributed to this worm. On the other hand Porter (1940) found no clinical evidence of injury nor weight loss resulting from infections. Symptoms which have been recorded are wasting, incoordination, weakness. The serious clinical cases recorded may have been related to lactation periods, nutritional deficiencies, or other disease conditions.

DIAGNOSIS

It is doubtful if a diagnosis of stomach worm disease can be made other than by necropsy. Determination of the presence of stomach worms can be made by culture of eggs in feces and examination of resultant larvae

TREATMENT

One of the only critical studies done on this species was that of Bozicevich and Wright (1935). They recommended administration of carbon disulphide either by stomach tube or capsule to animals fasted 30 to 48 hours prior to treatment. The suggested dose was 0.1 cc. per kilogram of body weight or 1–5 cc. per 100 pounds of body weight. This dose in their hands proved to be about 86 per cent efficacious.

PREVENTION

Porter (1939) indicated that strict adherence to the sanitation system produced pigs with lower incidence and lower percentage of heavy infections. Alicata (1935) experimentally showed that infective larvae could neither withstand −20° C nor 9 hours nor drying for 210 minutes. From these data one might suspect that *Hyo-*

strongylus larvae cannot withstand over wintering in the northern states

Ascarops strongylina — Physocephalus sexalatus — Thick Stomach Worms

Family Spiruridae. Mouth usually with tri lobed lateral lips. Sometimes lips are absent or small ventral or dorsal lips present. A chitinized cylindrical vestibule usually found behind the mouth. Esophagus long divided into an anterior muscular and posterior glandular portion. Cervical papillae present. Male caudal alae well developed and supported by pedunculated papillae of which there are usually 4 preanal pairs. Female vulva near middle of the body, oviparous

HOSTS

The adult is found in the stomach of swine. *Physocephalus* has the ability to encyst in the stomach of a number of hosts and remain dormant until eaten by swine

DISTRIBUTION

Both of these worms have a very wide distribution, appearing in Africa, Asia, Australia, Europe, and North and South America. Incidence figures seldom have been given. Spindler (1912) indicated that the incidence of these worms in swine in certain midwestern states has been found to be as high as 90 per cent of the animals examined. In the same report a 50 to 80 per cent incidence was recorded in the southeastern states. On the other hand the writer has never encountered these two worms in necropsy examinations of pigs in Michigan

ORGANS AND TISSUES INVOLVED

The mucosa of the stomach appears to be the only tissue and organ involved

MORPHOLOGY

Ascarops strongylina. Small red-colored worms. A narrow cuticular wing is found on the left side running from a point about 3 mm from the anterior end and ending about 2 mm from the posterior end below the lips and projecting into the buccal

cavity are two chitinous teeth formed by a prolongation of the wall of the pharynx The pharynx is marked by a series of chitinous ridges forming a continuous spiral

Male is 10-15 mm long possessing bursal wings or alae These wings extend to the posterior tip with the right one about twice as wide as the left Five pairs of somewhat asymmetrical stalked papillae support the bursa Spicules unequal the left being 2 24-2 95 mm long and the right 457-619µ long

Female is 16-22 mm long Vulva slightly anterior to the middle of the body Anus 215-275µ from the caudal tip

Eggs oval 34-39µ long by 20µ wide with thick shells Embryos well developed in the shell before oviposition

Physocephalus sexalatus Small red colored worms Head with two tri lobed lips Head is marked off from body by a cuticular inflation ending in a circular de marking margin just anterior to the pos terior end of the pharynx The pharynx contains a spiral band of 21 to 25 turns sometimes broken into discrete bands There are three lateral cuticular wings starting at the base of the cephalic inflation and extending about one third of the body length

Male is 6-9 mm long Narrow bursal membranes are present which are sup ported by 4 pairs of long stalked preanal papillae Postanal papillae (4 pairs) are near the tip of the tail and short The entire male tail is twisted about three turns Spicules unequal, the longest being 2 1-2 25 mm, the shorter one 300-350µ long

Female 13-19 mm long Anus 120µ from the caudal end Vulva is posterior to the middle of the body

The eggs are 22-26µ wide by 41-45µ long and slightly flattened at the poles Embryos are well developed in the shell prior to oviposition

LIFE CYCLE

The life cycles of these two worms are apparently quite similar Various species of dung beetles consume the eggs The larvae hatch and develop in the body cav

ity within a cyst to the third infective stage According to Alicata (1935) this may take 28 days or longer in the case of *Ascarops* and 36 days or longer for *Physocephalus* It is known that both species of stomach worms are capable of utilizing transport hosts but little is known of their actual parasitic development in the stomach of swine

LESIONS AND CLINICAL SIGNS

Foster (1912) indicated from the work of others that a pseudomembrane may be formed at the pyloric end of the stomach under which these worms are found partly attached to the stomach wall Red patches the size of a penny appeared around the pin prick opening made by the worm It has been suggested that the membrane is formed when the worms inoculate the mucosa with *Spherophorus necrophorus* bacilli which are commonly present in the stomach It has also been indicated that the worms may produce a gastritis and small ulcerations There are probably no pathognomonic symptoms present to aid in diagnosis

DIAGNOSIS

Characteristic eggs in the feces and necropsy examination appear to be the only way to tell if this infection is present

TREATMENT

There is very little experience on record due to the failure to diagnose cases while alive however Boncevich and Wright (1935) found that carbon disulphide given by capsule or stomach tube was effective against *Ascarops strongylina* when used after fasting 36 to 44 hours at the level of 0 1 cc. per kilogram of body weight No data have been recorded for *Physocephalus*

PREVENTION

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Stephanurus dentatus – Kidney Worm of Swine

Family Strongylidae   As given under *Oesophagostomum*

(1935) found they could develop to maturity in the guinea pig in about 19 days, although no mention of "egg-laying maturity" in this host was noticed.

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HOSTS

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DISTRIBUTION

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ORGANS AND TISSUES INVOLVED

The mucosa of the stomach appears to be the only tissue and organ involved.

MORPHOLOGY

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others Those that find their way to the perirenal tissues form cysts with fistulas leading into the ureters providing for dissemination of eggs The time necessary for development from egg to egg is said to be a minimum of 6 months, however, some think it may be several times that Kidney worm disease is most frequently found in pigs much older than 6 months As has been remarked by Spindler and Andrews (1955), the highest incidence of the parasite determined by eggs in the urine has been in animals 6 to 7 years old These authors also mentioned that their experimental infections with production of eggs during the patent period have not been accomplished Tromba (1955) has demonstrated experimentally that earthworms (*Eisenia foetida*) can be infected by exposure to third stage larvae and after 4 days they may be found in the brown bodies of the earthworm Swine fed these earthworms developed typical kidney worm liver lesions, although Tromba was not able to develop patency in the pigs up to 9 months after infection This lends credence to the possibility that some steps or variables in the life cycle are yet to be established

LESIONS AND CLINICAL SIGNS

In noting lesions, one might well include many organs of the body, however, these are not constant Certainly the liver lesions come as close to constancy as any White fibrotic spots, usually larger than those produced by *Ascaris*, often form streaks due to their coalescence and may be noted on the surface of the liver In heavy infections one may find the total liver changed in color from red to reddish gray

In cases where skin infection prevails, discrete nodules may appear on the skin If they are numerous, edema of the area may be present After a time a yellowish crust may form on the nodule There may also be a palpable swelling of local lymphatic glands

Oddly enough, little attention has been called to lesions of the kidney proper Ross and Kauzal (1932) called attention to necrotic infarcts in the kidneys of some cases Spindler and Andrews (1955) re-

marked that occasionally mature worms are found in the kidney proper

There are no clear cut symptoms associated with kidney worm disease The worms seem to manifest themselves differently in pigs, depending somewhat on the age of the host In heavily infected young pigs fatal cachexia may result If the infection is fairly light, the pigs may show faulty feed utilization and develop an emaciated condition This may be due to interference with liver function Posterior paralysis has been reported occasionally, caused perhaps by migratory forms of the worm in the central nervous system Most authorities list emaciation or unthriftiness as a prevailing symptom, but most parasitic afflictions can produce such symptomatology

DIAGNOSIS

Necropsy examination or the presence of characteristic eggs in the urine offers the only sound approach at present. It is very unfortunate too, since young animals may have heavy infections in the migratory phase and never be diagnosed

TREATMENT

There is no known treatment

PREVENTION

Some hope is offered here if the problem is serious enough to warrant the expenditure of much time and effort It has been generally recommended that fence lines and paths be kept free of vegetation so as to permit maximum drying Swine defecate and urinate most frequently along fence paths and near feeders One end of the pasture is kept free of vegetation and the feeders and waterers are placed here. Waterers should be of a design to prevent spilling or leaking If young pigs are in the pasture with the sow, creep feeders may be used to prevent contact of the young pigs with the sow feeder where a higher concentration of infective larvae will most likely be found The whole program rests on obtaining maximum sunlight and dryness on the areas of highest infectivity.

HOSTS

Normally the host is swine The ox and donkey have been recorded as hosts of this species but probably are incidental and of no significance

DISTRIBUTION

S dentatus is most prevalent in the south Atlantic and south central states It has been reported from a number of states farther north Spindler and Andrews (1955) stated that lesions due to kidney worms have been seen in swine originating from Massachusetts, Kansas, Nebraska, and central Washington No doubt there are other reports from northern states, but some, if not all, of these reports relate to swine shipped interstate At any rate, kidney worm disease has not yet reached serious proportions in any of the northern states In general the kidney worm has a wide geographical distribution including Ghana, Natal, Annam, Java, Sumatra, Australia Philippines, Hawaii, the West Indies, and Brazil It seems to be limited only by climatic conditions

ORGANS OR TISSUES INVOLVED

Since this nematode has a migratory phase one might list almost any organ as a possible site Actually, the prominent places where they are to be found are the liver and the fat surrounding the kidneys and ureters Actually, larval forms have been located in the brain, spinal cord, musculature, lungs, mesenteries, and pancreas Since part of the larval migration is carried by the blood stream, one may find larvae lodged in unusual places Whether they would continue their cycle without recourse to the liver is open to question

MORPHOLOGY

These are rather thick, robust worms, the males measuring 20–30 mm in length by 1 2 mm maximum thickness Females measure 25–45 mm in length and up to 1 8 mm thick The buccal capsule is about 0 18 mm wide and deep There are usually 6 teeth, variable in shape, at the base of

the capsule The leaf crown has about 50 small elements There is a club-shaped esophagus about 1 6 mm long in the female The nerve ring is found about 0 5 mm from the anterior end and the excretory pore is 0 5–0 6 mm behind the nerve ring

The spicules, slightly swollen at the tips, have transversely striated alae Spicules measure 0 66–1 0 mm, being either equal or unequal A flattened heart shaped accessory piece is present and measures about 0 075 mm in length

The female tail is about 0 59 mm long Near the level of the anus is a pair of globular formed processes The vulva is 1 36 mm from the anus

Eggs are quite large, strongyle like, measuring 90–115μ long by 43–65μ wide

LIFE CYCLE

Characteristic eggs are eliminated with the urine in the early stages of segmentation A million eggs have been known to pass from one hog in a day Given optimum conditions, the eggs develop larvae and hatch in 24 to 48 hours These larvae become infective within 3 to 5 days thereafter

The infective state is quite vulnerable to changes in temperature, direct sunlight and unusually dry conditions If experimental conditions are well controlled the larvae may survive nearly 3 months It is the opinion of some that they may last longer than that under field conditions (Spindler and Andrews, 1955)

Swine may become infected either by oral route or by skin penetration It is the consensus that, once the larvae are in the pig, the portal circulation serves to convey the larvae to the liver They may stay in the liver several months, growing and migrating through the organ These movements, needless to say, cause great damage and scarring on the liver The developing worms in the liver finally break through the liver capsule and enter the body cavity From here to the kidney many larvae may be lost in other accessible tissues such as pancreas spleen, mesentery, muscles, and

well as petechial hemorrhages in the lungs, heart, and intestinal mucosa. Pericarditis has been seen and third stage larvae have been recovered from the musculature, myocardium, tongue, brain, spinal cord, and lungs.

While there appear to be no pathognomonic symptoms, pigs may be restless and irritable, have anorexia, reduced growth rate, diarrhea, vomiting, and intestinal hemorrhage, and even may die.

DIAGNOSIS

Since many swine may shed some of the characteristic eggs and yet not have clinical disease and since nothing is known about the relationship of egg levels to the disease, it is doubtful if diagnosis can be accomplished prior to necropsy. Large numbers of typical eggs combined with the rather vague symptomatology offers some promise of diagnosis.

TREATMENT

Nothing is used in swine at the present time.

PREVENTION

Prevention is centered around current sanitation measures and the selection of a dry, unshaded area as a swine lot.

Trichuris suis — Whipworm

Family Trichuridae: Medium to large worms, the anterior (esophageal) part of the body may be longer or shorter than the posterior. The posterior body may be thicker or only slightly thinner than the anterior. Mouth simple. *Male:* spicule single or rarely with only a copulatory sheath. *Female:* vulva near termination of esophagus. Oviparous with thick-shelled eggs, which are barrel-shaped with plugs at each end and are deposited with an unsegmented ovum

HOSTS

Known hosts are pig, man, wild boar, and monkey.

DISTRIBUTION

T. suis is cosmopolitan where swine are raised.

ORGANS OR TISSUES INVOLVED

The cecum, colon, and appendix (man) are the organs involved.

MORPHOLOGY

The male is 30–45 mm. long and 0.45–0 65 mm. maximum thickness. The esophageal portion is about two-thirds to three-fifths of the total length (Fig. 28.6). The spicule measures 2–3.35 mm. long and 0.056 mm. maximum thickness. Its tip may be round or pointed. The ejaculatory duct is about 2.9–3.4 mm. long, which is about half, or sometimes much less than half, as long as the vas deferens with which it is connected by a narrow duct 0.5 mm long The testis is closely convoluted throughout its length.

In the female the vulva is not prominent Eggs measure 50–56μ by 21–25μ wide. Eggs are colorless when layed, but in the feces they assume a dark reddish brown color

LIFE CYCLE

Eggs are passed unembryonated. The egg develops to the infective stage in 1 to 12 months depending on environment When ingested by the proper host, the larvae hatch and develop to maturity in the cecum or colon in about a month.

FIG. 28 6—Whipworms, Trichuris suis. Note the attenuated anterior end.

There have been several chemicals recommended for treating the soil and the flooring of pens Methyl bromide is effective but costly and of little value in pastures or over large areas Some borate salts of sodium have likewise been useful as means of control although their use has been limited because of their destruction of vegetation

The fact that kidney worm eggs are seldom expelled from infected animals until they are two or more years old implies that the continuity of this disease depends on carry-over of breeding stock An early replacement of breeding stock with younger animals may be very helpful in controlling kidney worm disease

Strongyloides ransomi — Intestinal Threadworm of Swine

Family Rhabditidae Small forms free living or parasitic or with both free living and parasitic phases Three sided prismatic or tubular buccal cavity usually without teeth Esophagus usually with a posterior bulb containing valves and frequently also with a prebulbar swelling Reproductive organs simple Oviparous sometimes parthenogenetic or hermaphroditic

HOSTS

The pig is the only known host of *S ransomi*

DISTRIBUTION

There are some species of this genus found in swine on a cosmopolitan basis *Strongyloides ransomi* is thought to be an American form while *S suis* has been accepted as European Actually there have been few observations of this tiny nematode genus on a geographic basis in the United States Schwartz and Alicata (1930) concluded from examination of specimens from Moultrie Georgia that *S suis* probably occurred in the United States however, most authors refer only to *S ransomi*

ORGANS AND TISSUES INVOLVED

Since these are skin penetrating forms and have a blood migration one might expect to find larvae in many of the principal organs Organs that have been singled out are skin, lungs heart and intestine

MORPHOLOGY

No parasitic males have been described but the parasitic females are tiny 3.33–4.49 mm long and 54–62μ wide The esophagus is 605–883μ long by 47μ wide The anus is located 53–83μ from the tip of the tail The transverse vulva with protruding lips is posterior to the middle of the body but at a distance of 1.1–1.6 mm *from the tip of the tail*

The eggs are ellipsoidal thin shelled containing an embryo They measure 45–55μ long by 26–35μ wide For a description of larvae, consult Schwartz and Alicata (1930)

LIFE CYCLE

The small embryonated eggs are passed in the feces At room temperatures they hatch in 12 to 18 hours The resultant rhabditiform larvae may develop into filariform or infective larvae 22 to 24 hours after the larvae hatch from the egg The infective larvae are capable of penetrating the skin and proceeding to the lungs via the blood stream and thence from the alveoli of the lungs to the bronchi esophagus, stomach and small intestine where they become adults about 7 days after infection

It has been shown by Lucker (1934) that oral ingestion of infective larvae can produce infection also The exact fate of these orally ingested larvae needs further study Whether the infective larvae in this case make a lung migration or remain in the intestinal mucosa requires more documentation

As with other species of *Strongyloides S ransomi* is capable of developing a free living generation of adult males and females which in turn develop infective parasitic larvae

LESIONS AND CLINICAL SIGNS

A number of lesions have been described Skin eruptions following percutaneous infection have been noted as

demonstrated by muscle presses or by digestion of muscular tissue in an acidified pepsin solution and examination under the microscope.

TREATMENT

No treatment is known.

PREVENTION

Prevention is accomplished by several means. Studies have shown the highest incidence of *Trichinella* in garbage fed swine (1–6 per cent), while in grain-fed animals it has been less than 1 per cent. Emphasis has been placed on cooking garbage or not feeding it. Public education concerning the importance of properly cooked pork products has also been somewhat effective. Trichinosis is primarily a public health problem since its damage to swine is minimal.

ACANTHOCEPHALIDS

Macracanthorhynchus hirudinaceus—Thorny-headed Worm

Phylum **ACANTHOCEPHALA:** Endoparasitic vermiform organisms without a digestive tract but possessing an invaginable hook-armed proboscis as its organ of attachment.

Family Oligacanthorhynchidae: Worms of considerable length, slightly ringed bodies being frequently curved or coiled. Proboscis short, ovoid, or globular and armed with a few circles of hooks decreasing basally

HOSTS

Hosts of *M. hirudinaceus* are swine, wild boar, and occasionally dogs and monkeys. There have been early reports of human infections, but there is doubt that these actually occur.

DISTRIBUTION

This worm is cosmopolitan where swine are raised.

ORGANS OR TISSUES INVOLVED

According to Kates (1944), the worms are generally located in the jejunum 23 to 43 feet from the pylorus.

MORPHOLOGY

Large nematode like worms superficially resembling ascarids They appear to have wrinkling or transverse pseudosegmentation. The proboscis, usually penetrated into the intestinal wall (Fig. 28.7), bears five of six rows of recurved spines. Females measure 20–65 cm. long by 4–10 mm. wide. Males are 5–10 cm long and 3–5 mm. wide They bear a bell shaped, bursa like structure at their terminal end.

Eggs measure 80–100µ long by 56–65µ wide. The egg contains a larvae when layed.

LIFE CYCLE

Characteristic eggs pass out with the feces where they are eaten by beetle grubs of the genera *Cotinus* and *Phyllophaga* The larvae within the egg hatch in the beetle midgut and the larvae migrate to the body cavity, where they develop to the

FIG. 28.7—Thorny-headed worms, *Macracanthorhyncus hirudinaceus*.

LESIONS AND CLINICAL SIGNS

In swine there is little record of lesions or symptoms

DIAGNOSIS

There is no diagnosis of the disease reported, but the presence of the worms can be detected by their characteristic eggs in the feces

TREATMENT

In swine no treatment is prescribed, although feeding skim milk has a tendency to flush some worms out of the host

PREVENTION

The standard swine sanitation system seems to give some control over this species of worm

Trichinella spiralis

Family Trichinellidae   Small worms, simple mouth Male spicule and copulatory sheath absent. *Female* vulva in esophageal region Viviparous Parasites of mammals adults in intestine and larvae in muscles

HOSTS

The hosts are primarily man and pig although *T spiralis* has been reported in ox, sheep horse, dog cat, rabbit rat, and many other mammalian hosts It has been reported in such wild mammals as the polar, brown, and grizzly bears, arctic fox and white whale

DISTRIBUTION

T spiralis is cosmopolitan where swine are raised

ORGANS OR TISSUES INVOLVED

Primarily skeletal muscles are affected although lesions may be found in the myocardium, lungs, and occasionally the brain and meninges

MORPHOLOGY

Male is 1 4–1 6 mm in length and about 0 01 mm thick The female is 3–4 mm in length and 0 06 mm thick At the posterior end of the male there is a pair of ventrally directed conical processes located at the sides of the cloaca, and between the processes are two pairs of papillae In the female the vulva is near the middle of the esophageal region of the body

The embryos on hatching measure 0 09 –0 16 mm in length and 6–9μ in thickness

LIFE CYCLE

Copulation takes place in the small intestine of the host The female burrows into the mucous membrane by way of the glands of Lieberkuhn and makes its way to the lymph spaces Large numbers of embryos are deposited which enter the lymphatic and blood streams and are carried all over the body On reaching desirable muscles, the larvae penetrate the sarcolemma of the muscle fibers Here the larvae become enclosed in cysts, usually one to a cyst although as many as seven have been recorded in a single cyst The time required to reach the muscles varies from 8 to 25 days after infection Viable cysts may remain intact for years although a process of calcification begins gradually to destroy the larvae and capsule

The infection is passed on to man or other mammals by ingestion of uncooked or improperly cooked pork products

LESIONS AND CLINICAL SIGNS

Pigs are usually quite tolerant to the parasite There is very little effect on the gastrointestinal tract and the main lesions are those of the larvae in the musculature For an exact description of cellular changes in the muscle, one may refer to Gould (1945)

Symptoms in pigs have been produced experimentally but are seldom, if ever seen in natural infections There may be a loss of appetite, colicky pains, paralysis of the hind quarters, incontinence of urine and feces diarrhea, stiffness of the muscles and itching

DIAGNOSIS

This disease as a clinical entity has probably never been diagnosed in living swine At necropsy, the larvae are easily

conclude that the Cincinnati report was one of most unusual circumstances Kern kamp (1946) did not even include *Para gonimus* in his discussion of parasites and parasitic diseases of swine

There is no known treatment of domes tic animals

Fasciola hepatica

Normally *Fasciola hepatica* is a parasite of sheep and cattle and in some countries even man In the United States this para site is a rare one in swine, and the litera ture is scarce It is enough to say that animals acquire the infection by ingesting the metacercariae encysted on grass The fluke is a large parasite inhabiting the liver and bile ducts No serious outbreaks seem to have been reported in swine although there is voluminous literature about it in other hosts It is of interest that in for eign lands that there is enough fascioliasis in swine to merit treatment tests Winter halter and Delak (1956) have developed a treatment in swine by use of subcutane ous injections of carbon tetrachloride mixed with liquid paraffin

In the United States, particularly in the south and western coastal plain, care might be taken not to pasture swine on areas used by sheep or cattle

Taenia solium —
Cysticercus cellulosae — "Measly Pork"

This tapeworm parasite occurs in man as an adult and occasionally as a larval form It occurs in swine in the larval form only These larvae may be seen in the muscles of the heart, tongue, diaphragm, and, in fact, almost any body muscle They are tiny white, lemon shaped bladders about ½ inch long and ¼ inch wide with a protuberance on one side which is the invaginated scolex, or organ of attachment. They apparently cause little discomfort to the pig and no clinical evidence of disease

Swine receive the infection by ingesting eggs disseminated by careless human habits. With the improvement of rural sanitation in the United States, the incidence has steadily dropped Schwartz (1952) stated

that federally inspected meat showed 10 to 26 cases out of 45 to 59 million hogs slaughtered It is increasingly difficult to obtain either the adult or bladderworm larval stages for teaching purposes Thor ough cooking of pork or freezing tempera tures for a week destroy the larvae

Taenia hydatigena
Cysticercus tenuicollis

The adult tapeworm lives in the in testine of dogs and related carnivores This bladderworm or larval stage is most often found in sheep and cattle, although some times in swine

Domestic animals acquire their infection by ingesting the eggs from the droppings of infected dogs The eggs hatch in the intestine and the embryo finds its way to the liver via the blood stream After reach ing the liver it usually burrows out and may be found on the surface of the liver or, more commonly, attached to the omen tum or organs in the peritoneal cavity Dogs must ingest the bladderworm to com plete the cycle The larva is usually found in a cyst filled with fluid, the larval cyst is very large, measuring several inches in di ameter when mature

Usually there are no clinical signs in pigs or other animals although Anthony (1955) indicates that death can ensue in very young pigs

Control measures involve treating dogs for the adults and preventing the inges tion by dogs of the infective larval stage No treatment is known and diagnosis can be made only at necropsy

Echinococcus granulosus — Hydatid Disease

Of the tapeworm parasites of swine, the larval stages of this one probably consti tute the major cestode problem of swine

The adult of this species is a very tiny worm composed of a scolex (head) and three or four segments A dozen or more entire adult worms could be placed under one microscope slide coverslip The adult lives in the small intestine of dogs and related carnivores which disseminate, with their excrement, the eggs of the tapeworm on pastures and swine lots

infective stage after about 3 months in the grub

Pigs acquire the infection by ingesting grubs containing infective larvae The time element for development to maturity in the pig is about 3 to 4 months

This infection has been reported in man in the Volga Valley of Russia and it was stated that beetles of the genus *Melontha* were eaten raw The record was made years ago and no recent reports have substantiated it.

LESIONS AND CLINICAL SIGNS

When the proboscis or hook bearing snout of the parasite penetrates the mucosa an inflammatory area may be established As the thorny headed worm moves from place to place the wounds resolve into pealike nodules often filled with caseous material It has been said that perforation of the intestine may occur Should this happen any leakage of intestinal contents would most certainly set up a peritonitis which could become fatal

There are no specific symptoms and it is unusual to find large numbers of worms per pig although these parasites probably contribute to unthriftiness of infected animals

DIAGNOSIS

Diagnosis can be made only by the presence of eggs in the feces or at necropsy

TREATMENT

No treatment is known

PREVENTION

Pastures kept free of litter (boards trash leaves) may reduce the grub population Actually good feed and enough of it will help keep rooting to a minimum and the ringing of swine may help break the relationship of swine to the grub The eggs of the thorny headed worm are very resistant and may remain infective to the grub for several years on pasture

TREMATODES AND CESTODES

The group of parasites comprising the trematodes or flukes the tapeworms and

their intermediate stages are of minor importance to those interested in swine diseases In many cases the pig is not the normal host but rather an accidental host Incidence records of these parasites in swine are very limited and probably quite inaccurate It is recognized that a number of different species of flatworms have been reported from swine Hall (1933) listed 18 trematodes noting that the flukes are seldom of material economic importance in swine husbandry He further indicated that there were no adult tapeworms normally existing in swine

Phylum **PLATYHELMINTHES** These are bilaterally symmetrical, soft bodied flatworms lacking a blood vascular system and true coelom These worms possess a unique excretory system utilizing flame cells All but one class are parasitic.

Paragonimus kellicotti

This trematode is a fleshy, spinous worm of fairly large size ($\frac{1}{2}$ to $\frac{3}{4}$ inch long) living as an adult in the lung of many different hosts Pigs are probably accidental hosts Mink seem to be the common host in the United States although it may be found in dogs cats sheep goats and even man

The host acquires the infection by ingesting the metacercarial stages found in the body of crayfish A small snail *Pomatiopsis lapidaria*, appears to be the first intermediate host in the United States The ingested metacercaria penetrates the intestinal wall and wanders to the pleural cavity enters the lung and encysts There are a local inflammatory reaction adhesions and even fibrosis Large brown operculate eggs are coughed up and may be demonstrated in the sputum or feces of the definitive host The eggs measure 78–96μ long by 18–60μ wide They are in the single-celled stage when passed

The distribution in swine has not been well recorded Many texts refer to a report by Stiles and Hassall (1900) from Cincinnati From the lack of further reports on this parasite in swine one may

Allen, R W 1945 Trials with sodium fluoride as an ascaricide for swine No Amer Vet. 26 661
——— and Jones, L D 1946 The efficacy of sodium fluoride in removing ascarids of swine No Amer Vet 27 358

Andrews, J S 1956 Animal Diseases Yearbook of Agr , U.S.D.A

Anthony, D J 1952 Diseases of the Pig 4th ed Williams and Wilkins Co , Baltimore

Baylis H A 1936 Fauna of British India (nematodes) I 36

Beaver, P C 1954 Parasitic diseases of animals and their relation to public health Vet Med 49 199

Betts L O 1954 Ascaris lumbricoides as a cause of pneumonia in pigs Vet Rec. 66 749

Bozicevich, J and Wright, W 1935 Carbon disulphide for the removal of stomach worms from swine Vet Med 30 390

Burch, G R , and Blair, H E 1955 A new ascaricide for swine. Jour Amer Vet Med Assn 126 504

Chandler A C 1955 Introduction to Parasitology 9th ed John Wiley and Sons New York

De Boer A 1935 Experimentelle Untersuchungen uber Ascaris lumbricoides des Menschen und des Schweines Zeitschr f Infektionskrankh parasit Krankh u Hyg d Haustiere 48 248

Dougherty, E C 1944 The genus Metastrongylus Molin 1861 (nematoda Metastrongylidae) Proc Helm Soc. Wash 11 66

Dunn, D R 1956 Studies on the pig lungworm (Metastrongylus spp) II Experimental infection of pigs with M apri Brit Vet Jour 112 327
——— Gratius M A and White, E G 1955 Studies on the pig lungworm (Metastrongylus spp) Observations on natural infection in the pig in Great Britain Brit Vet. Jour 111 271

Enzie, F D and Colglazier, M L 1955 Present day trends in anthelmentics Proc Book Amer Vet Med Assn p 153
——— Habermann R T and Foster A O 1945 A comparison of oil of chenopodium phenothiazine and sodium fluoride as anthelmentics for swine Jour Amer Vet Med Assn 107 57

Foster, W D 1912 The roundworms of domestic swine with special reference to two species parasitic in the stomach U.S.D.A Bur Anim Ind Bull 158

Gooden, T 1921 The anatomy of Oesophagostomum dentatum (Rud) a nematode parasite of the pig with observations on the structure and biology of the free living larvae Jour Helm 2 1
——— 1926 Some stages in the development of Oesophagostomum dentatum from the pig Jour Helm 4 191

Gould S E 1945 Trichinosis Charles C Thomas Springfield Ill

Guthrie J T 1954a Critical tests with cadmium anthranilate as an ascaricide in swine Vet Med 49 413
——— 1954b Further observations on the efficacy of cadmium anthranilate as an ascaricide in swine Vet Med 49 500
——— 1956 Critical tests with piperazine as an ascaricide in swine Vet Med 51 235

Habermann, R T, Enzie, F D, and Foster, A O 1945 Tests with fluoride, especially sodium fluoride as anthelmintics for swine Amer Jour Vet Res 6 131

Hall, M C 1933 Internal parasites of swine Vet Med 28 26

Hassall A and Stiles, C W 1892 Strongylus rubidus, a new species of nematode parasitic in pigs. Jour Comp Med and Vet Arch 13 207

Hobmaier, A, and Hobmaier M 1929 Die Entwicklung der Larve des Lungenwurmes Meta strongylus elongatus (Strongylus paradoxus) des Schweines und ihr Invasionsweg sowie vorlaufige Mitteilung uber die Entwicklung von Choerostrongylus brevuvaginatus Münch tierarztl Wschr 80 565

Kates, K C 1941 Observations on the viability of eggs on lungworms of swine. Jour Parasit 27 265
——— 1944 Some observations on experimental infections of pigs with the thorn headed worm Macracanthorhynchus hirudinaceus Amer Jour Vet Res 5 166

Kelley, G W Olson, L S, and Garwood, V, 1956 A field evaluation of ascaricides in swine Vet Med 51 97

Kennedy, P C 1954 The migrations of the larvae of Ascaris lumbricoides in cattle and their relation to eosinophilic granulomas. Cornell Vet 44 531

Kernkamp, H C H 1916 Parasites and parasitic diseases of swine Vet Med 41 315

Koino, S 1922 Experimental infection on the human body with ascarides. Jap Med World Tokyo 2 317

Leiper, J W G 1954 The piperazine compound V 19 for the removal of Ascaris and Oesophagostomum from the pig Vet. Rec. 66 596

Lindquist, W D. 1957a A low level piperazine study on pigs experimentally infected with Ascaris lumbricoides Amer Jour Vet Res. 18 119
——— 1957b A low level piperazine study on pigs naturally infected with Ascaris lumbricoides Amer Jour Vet Res. In Press.

Lucker, J F 1934 Development of the swine nematode Strongyloides ransomi and the behavior of its infective larvae. U.S.D.A Tech Bull 437

Once these eggs are ingested by sheep, cattle, swine, or even accidentally by humans the embryo escapes from the egg, burrows through the intestinal wall, and enters the blood stream where it is most often swept to the liver and lungs to develop into its cystic or hydatid stage Occasionally the cystic stages may be found elsewhere in the body, e g , the spleen, kidney, and rarely in the musculature The cysts in swine are usually the simple uniocular variety and may be fertile or sterile They vary in size from 1 cm to 7 5 cm in diameter Sometimes they may be so numerous as to cover the organ completely

The source of infection for swine is not definitely known Infected dogs can disseminate the eggs on swine lots but wild carnivores may also play a part In the South, where most of the incidence reports from the United States have been made, swine are known to range unconfined more often than in the northern states This factor might implicate wild carnivores as well as dogs

The problem of hydatid disease in swine cannot be easily circumscribed due to a paucity of incidence reports in recent years One may go back before 1900 (Stiles, 1898) and find references to 117 swine cases out of 2 000 hogs slaughtered in New Orleans A much larger survey apparently made about the same time, quoted by Magath (1937) was one case in 24 000 In the same publication, figures given from Nashville, Tennessee, for a 5 month period in 1936 were 0 64 per cent of 62 399 hogs slaughtered Ward and Bradshaw (1956) report a 4 03 per cent incidence from 8,066 swine from five different areas of

south and central Mississippi In the same report, these authors mentioned that 1,157 swine shipped to their packing house in Mississippi from St Louis, Missouri, had no hydatids It would appear that this disease may be a localized entity at times and related in some way to husbandry Recent reports are few from northern states although older records are available from Canada

There has been discussion in the literature about the amazing fact that so few cases are recorded from dogs This has led to the increased interest in the sylvatic mode of infection, but little account has been taken of the fact that many veterinary necropsies are done without benefit of screening and illuminating the contents of the small intestine Without such a method it is doubtful if these tiny tapeworms would be observed unless in very large quantities Since the eggs of *Echinococcus* appear exactly like those of other *Taenia* tapeworms, there is no way to alert the veterinarian to the presence of this tapeworm There certainly is a possibility that our incidence in domestic dogs is higher than recorded and that we have missed them at the necropsy table

The problem of hydatid disease in swine is of public health significance but produces no discernible effect on the pig It is discovered at necropsy or slaughter Treatment is not available for swine, nor are there diagnostic methods simple enough to be practical Control of the disease in pigs rests upon proper husbandry, an thelmintic treatment of farm dogs, and restricted range of swine to minimize their contact with infections in wild animals

REFERENCES

ABDULRACHMAN, S AND JOE L K 1954 Morphological differences between *Ascaris* from man and pigs Doc Med géog et trop 6 312

ALICATA, J E 1933 The development of the swine nodular worm *Oesophagostomum dentatum* Jour Parasit 20 73

——— 1934 Observations on the period required for *Ascaris* eggs to reach infectivity Proc Helm Soc Wash 1 12

——— 1935 Early developmental stages of nematodes occurring in swine U S D A Tech Bull 489 pp 1–96

——— 1955 Effects of sodium borate on swine nodular worm *Oesophagostomum dentatum* infective larvae in soil Jour Parasit (Suppl) 41 50

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Protozoa*

The smallest members of the animal king dom are usually considered to be unicel lular These animals belong in the phylum PROTOZOA Thousands of species have been described in this group, most of which are not parasitic in habit About 40 of these species have been found associated with domestic and wild swine Compara tively few species of swine protozoa are definitely known to be pathogenic Some species appear to damage body cells only when predisposing factors are present, while other species found in swine seem incapable of causing disease Future re search may alter present day opinions re garding the so-called pathogenic and non pathogenic protozoa of swine

The taxonomic relationships of the phylum PROTOZOA vary with the view point of the taxonomist For this discus sion of swine protozoa the classification of Kudo (1954) will be followed Briefly stated, the protozoa of swine belong to the following classes and genera (only the more important genera found in swine in North America will be discussed in detail in this chapter)

Class I Mastigophora Genera include *Trypanosoma, Chilomastix, Giardia, Tri chomonas,* and *Tritrichomonas* These Mastigophora are characterized by their possession of flagella for the purpose of

locomotion Reproduction usually occurs by longitudinal division

Class II Sarcodina Genera include *Endamoeba, Endolimax* and *Iodamoeba* These Sarcodina move in a creeping (amoe boid) manner through the action of pseudopodia Reproduction is by simple division

Class III Sporozoa Genera include *Eimeria, Isospora, Babesia, Toxoplasma Eperythrozoon,* and *Sarcocystis* (status un certain) Sporozoa have a rather complex life cycle with both motile and nonmotile forms Infections of host cells may take place by means of sporozoites either in gested or inoculated by an intermediate arthropod host. Reproduction is by simple division or through a series of sexual and asexual stages

Class IV Ciliata One genus, *Balantid ium,* is found in swine Locomotion is by means of many cilia on the surface Repro duction occurs by transverse division

The more important protozoa reported from swine will be considered under the name of the disease caused by individuals or by groups within the classes Nonpatho genic protozoa will be considered in certain of the groups Only a few species of proto zoa are host specific for swine

Trypanosomiasis

There are no pathogenic trypanosomes as yet reported from swine in North America Five species of trypanosomes have been

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MAGATH, T H 1937 Hydatid (Echinococcus) disease in Canada and the United States Amer Jour Hyg 25 107

MONNIG H W 1947 Veterinary Helminthology and Entomology 3rd ed Williams and Wilkins Co Baltimore

MORGAN, B B AND HAWKINS, P A 1949 Veterinary Helminthology Burgess Publ Co, Minneapolis

OTTO G F 1932 The appearance of unfertilized eggs of *Ascaris lumbricoides* Jour Parasit 18 269

PAYNE F K, ACKERT J E AND HARTMAN, E 1925 The question of the human and pig *Ascaris* Amer Jour Hyg 5 90

PORTER D A 1939 Effectiveness of the swine sanitation system in controlling swine stomach worms in the South Proc. Helm Soc Wash 6 21

——— 1940 Experimental infections of swine with the red stomach worm *Hyostrongylus rubidus* Proc Helm Soc Wash 7 20

RANSOM B H AND CRAM, E 1921 Course of migration of *Ascaris* larvae from the intestine to the lungs Anat Rec 20 207

——— AND FOSTER W D 1917 Life history of *Ascaris lumbricoides* and related forms Jour Agr Res 2 395

——— AND ——— 1920 Observations on the life history of *Ascaris lumbricoides* U.S.D.A Bull 817

REICHE P 1921 Über Askariasis der Schweine Inaug Diss (Berlin) Also in Deutsche tier arztl Wschr 30 420

ROSS I C AND KAUZAL, G 1932 The life cycle of *Stephanurus dentatus* Deising, 1839 the kidney worm of pigs Bull 58 Council for Sci and Ind Res Commonwealth of Australia

SCHWARTZ B 1931 Nodular worm infestation of domestic swine Vet Med 26 411

——— 1952 Parasitic diseases of swine transmissible to man Proc. Amer Vet Med Assn, 89th Ann Meet, p 385

——— AND ALICATA J E 1930 Species of the nematode genus *Strongyloides* parasitic in domestic swine Jour Amer Res 40 11

——— AND ——— 1934 Life history of lungworms parasitic in swine U.S.D.A Tech Bull 456

SHOPE, R E 1941 The swine lungworm as a reservoir and intermediate host for swine influenza virus II The transmission of swine influenza virus by the swine lungworm Jour Exper Med 74 49

SHORB D A 1948 Experimental infections of pigs with *Oesophagostomum dentatum* and *O longicaudum* Jour Parasit (Suppl) 34 26

SOLIMAN K N 1951 Observations on the orientation of certain lungworms in the respiratory tracts and on their feeding habits Brit Vet Jour 107 274

SPINDLER L A 1933 Development of the nodular worm *Oesophagostomum longicaudum* in the pig Jour Agr Res 46 531

——— 1938 Persistence of swine lungworm larvae in earthworms Proc Helm Soc. Wash 5 63

——— 1940 Survival of eggs of the swine ascarid in cultivated soil Jour Parasit (Suppl) 26 19

——— 1942 Keeping Livestock Healthy Yearbook of Agr U S D A

——— 1948 Ascarids a cause of loss to the meat industry through condemnation of swine carcasses due to icterus Jour Parasit (Suppl) 34 14

——— AND ANDREWS J S 1955 The swine kidney worm *Stephanurus dentatum* Proc 58th Ann Meet U S Livestock San Assn p 296

STEWART, F H 1916 On the life history of *Ascaris lumbricoides* Brit Med Jour 2 5

STILES, C W 1898 The inspection of meats for animal parasites Pt 1 The flukes and tape worms of cattle sheep and swine with special reference to the inspection of meats U.S.D.A Bur Anim Ind Bull 19

——— AND HASSALL H 1900 Notes on parasites The lung fluke *Paragonimus westermani* and its relation to parasitic hemoptysis in man 16th Ann Rep, Bur Anim Ind U.S.D.A P 560

STOLL, N R 1933 When are Ascaris eggs infective? Jour Parasit 20 126

SULLIVAN, J F AND SHAW, J N 1953 Incidence and effect of lungworm in Oregon swine Sta Tech Bull 28

TROMBA, F G 1955 The role of the earthworm *Eisenia foetida*, in the transmission of *Stephanurus dentatus* Jour Parasit 41 157

TURK R D AND HALE, J 1956 Observations on ascariasides in swine Jour Amer Vet Med Assn 128 405

WARD J W, AND BRADSHAW, R C 1956 New records of the occurrence of the hydatid tapeworm *Echinococcus granulosus*, in Central and South Mississippi Jour Parasit. 142 35

WHITE, E G 1955 The eggs of *Hyostrongylus rubidus*, Hall 1921 A stomach worm of the pig and their recognition in pig feces Brit Vet, Jour 111 11

WINTERHALTER, M AND DELAK, M 1956 Treatment of fascioliasis in pigs by subcutaneous injections of carbon tetrachloride Vet Arhiv 26 227

introduction of this group of parasites into swine

Trichomoniasis

There is little evidence that this condition exists *per se* It was first thought to be a cause of, or play a role in, necrotic enteritis of swine and, later, as a possible causative agent of atrophic rhinitis in swine In fact, in the latter disease, it is probable that the causative agent (s) or interaction of agents is unknown (Shuman *et al*, 1956)

The exact taxonomic relationship between the trichomonads of the nasal passage and the intestinal tract is not clearly understood Differences between them have been recorded but further work is necessary to determine whether these differences are sufficient to assign species status or not The lack of a marked host or organ specificity complicates this decision

MORPHOLOGY

Tritrichomonas suis (Gruby and Delafond 1843) is the name applied to the cecal form It is robust oval averaging $8.5 \times 6.8\mu$, with 3 anterior flagella of body length The undulating membrane filament continues as a trailing flagellum The axostyle has a short posterior tip

Tritrichomonas sp, found in the nasal cavity, is pyriform in shape and averages $13.3 \times 4.7\mu$ It contains the 3 anterior flagella and undulating membrane of the genus The axostyle has a short posterior tip with a chromatic ring

Tritrichomonas sp, found in the intestinal tract, is oval with an average length width measurement of $5.2 \times 3.5\mu$ The axostyle has a longer tip than the other two forms After culture these forms tend to resemble each other more closely Antigenic differences between *T suis* and *Tritrichomonas* sp found in the nasal cavity have been demonstrated by agglutination tests using rabbit sera

TRANSMISSION

Transmission is effected apparently by ingestion of the motile organism as a contaminant of feed or water

LESIONS AND CLINICAL SIGNS

There are no specific lesions or clinical signs attributable to natural infection with this organism

DIAGNOSIS

This is usually made from scrapings taken from the appropriate location in freshly killed swine Nasal swabs or washing will often reveal the organism in the living animal

TREATMENT AND CONTROL

At the present time there is no indication that treatment would be warranted

REMARKS

The transfer of trichomonads from the intestinal tract of swine to the genital tract of swine, goats, and cows, and the temporary establishment of *T foetus* in the uterus of swine introduces speculation as to the host specificity of these parasites The lack of success in the establishment of the infection in the preputial sheath of boars indicates that, at the present time, there is little possibility of this becoming a herd problem

Other Flagellates

Other flagellates reported from swine have primarily been parasites of man The
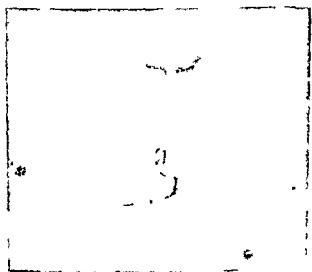
FIG   29 1 — Trypanosoma congolense   Blood smear   Approximately × 1,000

found in swine from other countries, principally from Africa, India and South America In view of the fact that the vectors and reservoir hosts for certain of these trypanosomes are available in North America it is important that these trypanosomes not be introduced from abroad

MORPHOLOGY

Trypanosomiasis is caused by members of the genus *Trypanosoma* These organisms have flattened, elongated bodies, tapering at both ends A long flagellum arises near the posterior end of the body and runs anteriorly on the surface, forming an undulating membrane, and continues as a free structure beyond the anterior end The trypanosomes vary in length according to the species and range from 9 to 35μ. Most of them are found in the blood and lymphatic systems of their hosts

TRANSMISSION

The trypanosomes are principally transmitted by blood sucking arthropods The more important arthropods involved include the tsetse fly, horse fly, stable fly, and reduviid bugs The trypanosomes multiply in these arthropods, and mechanical transmission can occur by blood inoculation The control of trypanosomiasis is based principally on attempts to destroy

the various arthropod vectors Treatment in swine is experimental and will not be reviewed Table 29 1 indicates the species recorded from swine

There are variations in the manifestation of this disease in domestic animals The predominant pathological change in trypanosomiasis is anemia due to the failure of erythropoeisis This may be accompanied by edema and diarrhea

DIAGNOSIS

The diagnosis of the disease is made by finding the characteristic trypanosomes in the blood stream (Fig 29 1), or, in the case of *T cruzi*, by finding leishmanial forms in tissue sections or smears from infected organs Other methods for diagnosis include precipitin tests, culturing, animal inoculation, etc

REMARKS

This disease, as yet not reported from swine in North America, represents a definite hazard to swine and a public health menace The report of Diamond and Rubin (1956) concerning *T cruzi* from raccoons in Maryland and the fact that swine may act as an important reservoir host for human infections of *T cruzi* (Faust, 1955) indicate that a continued vigilance must be maintained against the

TABLE 29 1
Trypanosomes Recorded From Swine

Name	Length	Vectors	Hosts	Location
Trypanosoma simiae Bruce 1912	12–24μ	Tsetse horse flies	Wart hog, monkey, sheep, goat	Africa
T congolense Broden 1904	8–21μ	Tsetse fly	Cattle sheep, goat, horse, donkey, camel, dog	Africa
T brucei Plimmer and Bradford 1899	12–35μ	Tsetse fly	All domesticated animals as well as many wild mammals	Africa
T gambiense von Jorde 1901	12 28μ	Tsetse horse, and stable flies	Primarily man, wild pig cattle, sheep, goat	Africa
T cruzi Chagas 1909	15–20μ 1 5–4μ*	Reduviidae	Man dog cat wood rat, opossum bat, raccoon, armadillo and other wild mammals	Central and South America

* Leishmani form

possible source of *E histolytica* infections in man, there have been five cases of *E polecki* reported from man Burrows and Klink (1955) believe that this infection may be more common than has been reported and that cases of uninucleated cysts have erroneously been diagnosed as *E histolytica*

Coccidiosis

There are five species of coccidia reported from swine in North America Four of the species belong in the genus *Eimeria* and one species belongs to the genus *Isospora*

MORPHOLOGY

These organisms go through a complicated life cycle involving both sexual and asexual reproduction Within the host body, they are confined to the epithelial cells of the digestive tract, where they undergo development through various stages depending upon the length of the infection These stages, in order of their appearance, are first the asexual trophozoite, schizont, and merozoite stages, then the sexual micro and macrogametocytes and the micro and macrogametes and, finally, the oocysts The oocyst stage is passed in the feces These oocysts vary in size from 9 to 29μ, in shape from oval to subspherical, and in color from colorless to brown, yellow, or pink, depending upon the species (Table 29 2) The oocyst passed in the fresh feces exhibits a double contoured wall and a single protoplasmic mass in the center This stage, in order to be infective to a new host, must complete its development outside the body The time required for this development varies from 4 to 12 days The *Eimeria* spp, when fully sporulated, show four sporocysts, each containing two sporozoites In the *Isospora* sp, fully sporulated oocysts exhibit two sporocysts with each containing four sporozoites

TRANSMISSION

This condition is transmitted by ingestion of sporulated oocysts as contaminants in food or water Cross transmission of coccidia between swine and other domestic animals has not been conclusively demonstrated

LESIONS AND CLINICAL SIGNS

This is a disease primarily of the young as adult animals have usually experienced the infection and become partially immune so that clinical signs are not shown The first sign of the infection is diarrhea which may be followed by constipation This diarrhea, in contrast to that of coccidiosis in cattle, is rarely bloody upon gross examination In addition emaciation, dehydration, and anorexia may occur The morbidity varies widely in different outbreaks (17 to 50 per cent) while the mortality remains rather low Recovered animals frequently show some stunting of growth and appear unthrifty The lesions are found mainly in the large intestine These lesions consist of congestion edema, and catarrhal and hemorrhagic inflammation of the intestine The small intestine may also be involved In experimental infections, the coccidia are confined to the

TABLE 29.2
COCCIDIA OF SWINE

Name	Size	Shape and Color	Sporulation Time
	(average)		(days)
Eimeria debliecki Douwes 1921	19 × 14μ	Smooth, oval, colorless	7 to 9
E scabra Henry 1931	29 × 20μ	Rough, oval, brown	9 to 12
E perminuta Henry 1931	13 × 11μ	Rough, subspherical, brown	11
E spinosa Henry 1931	19 × 14μ	Spiny, oval, brown	11 to 12
Isospora suis Biester 1934	22 × 19μ	Smooth, subspherical, pink	4 to 5

pathology and treatment of these forms have not been established. The following two flagellates are briefly described as a means of separating them from the previously discussed members of this class.

Chilomastix mesnili (Wenyon 1910) is an asymmetrical, pear-shaped organism with 3 anterior flagella and a cystostomal flagellum. It measures $10–24\mu$ in length in the trophozoite or motile stage. The oval-shaped cyst measures about 8μ.

Giardia lamblia (Stiles 1915) is a bilaterally symmetrical, pear-shaped organism (Fig. 29.2) which is convex on the dorsal surface, and the concave ventral surface forms a suction disc. The double nucleated trophozoite contains two axostyles and four pairs of flagella. The trophozoites measure $12–15\mu$ in length, and the oval cysts are $9–12\mu$ long. The cysts may contain either 2 or 4 nuclei.

Amoebiasis

Several species of amoeba have been reported from swine by fecal examination. They are: *Endamoeba histolytica, E. coli, E. polecki, Endolimax nana,* and *Iodamoeba butschlii.* Of these, apparently *E. polecki* should be considered as the amoeba of swine. From the reported cases of cysts, morphologically similar to *E. histolytica,* found in swine feces, it does not appear

that swine act as an important reservoir host for this infection of man. The following account concerns the amoeba *E. polecki* (von Prowazek 1912.)

MORPHOLOGY

The trophozoite stage is an irregular oval approximately $13 \times 16\mu$ and exhibits sluggish movements by pseudopodia. The nucleus appears large and pale. The cysts measure $4–17\mu$ (average 8μ) in diameter, are uninucleated, and may contain chromatoidal bodies.

TRANSMISSION

Amoebiasis is contracted by ingestion of the cyst in contaminated feed or water. The trophozoites are quite susceptible to environmental change and are rarely the transmitted form.

LESIONS AND CLINICAL SIGNS

This condition in swine rarely exhibits any clinical signs or lesions. Kinsley (1933) reported that pigs were gaunt, exhibited a temperature of 106 to 107° F., and had diarrhea. The necropsy findings were intestinal inflammation characterized by a milky exudate. Tissue sections reveal the amoeba to be in the intestinal lumen, in the crypts, or at the margins of ulcers. As amoebae have been reported from many apparently normal animals, the primary cause of the infection should be investigated and other causes of enteritis eliminated.

DIAGNOSIS

The characteristic uninucleated cysts are found in the feces. If the diarrhea is severe, the motile forms may be passed. To find this stage the specimen must be fresh and not allowed to cool. Smears may be made and stained with iron hematoxylin in order to facilitate finding the cysts or trophozoites. Because of the similarity of *E. polecki* and *E. histolytica,* only those typical cysts with four nuclei should be diagnosed as *E. histolytica.*

REMARKS

Although there is some disagreement concerning the exact role of swine as a
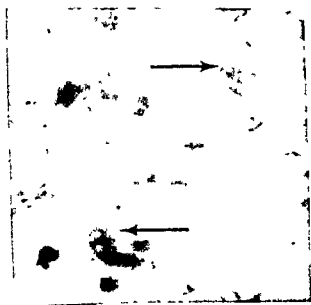
FIG. 29.2 — *Giardia lamblia.* Fecal smear. Approximately X 1,000.

FIG. 29.5 — Eimeria sp. Sporulated oocysts. Approximately X 700.

Control measures should include adequate clean water from fountains or troughs and racks or bunks for feed. Adequate space should be provided to avoid over-crowding. Pasture rotation to reduce the amount of contamination is helpful, but this must not be the only control practice used. Avery (1942) showed that swine coccidia may remain viable on soil for 15 months with the surface temperature ranging from 40 to $-4 5^{\circ}$ C, and will withstand freezing for at least 26 days.

REMARKS

Coccidiosis is usually a self limiting disease and the course lasts about two weeks. Low numbers of oocysts may be shed for longer periods of time and by partially immune animals.

FIG. 29 6 — Isospora sp. Sporulated oocysts. Approximately X 700.

Toxoplasmosis

This is a widespread disease which can be acute or chronic, symptomatic or asymptomatic. The causative agent, *Toxoplasma gondii* (Nicolle and Manceaux 1908), is apparently the same regardless of the host involved.

MORPHOLOGY

T. gondii presents a crescent or arc-shaped structure with one end more rounded and the other attenuated (Fig. 29.7). It measures about $4-7\mu$ in length and about $2-4\mu$ in width There is no centrosome or kinetoplast visible, and the nucleus appears as a mass of chromatin granules These proliferating forms are found in a wide variety of cells and organs of the body, including the lymph nodes, lungs, spleen, liver, and intestines In the chronic stage, the infection is apparently maintained by a cyst or pseudocyst which is confined to the brain or myocardium These pseudocysts are rather large and give the appearance of having a definite cyst membrane surrounding many toxoplasma bodies.
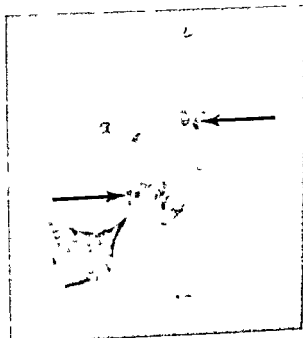
FIG. 29.7 — Toxoplasma gondii. Peritoneal exudate. Approximately X 1,000.
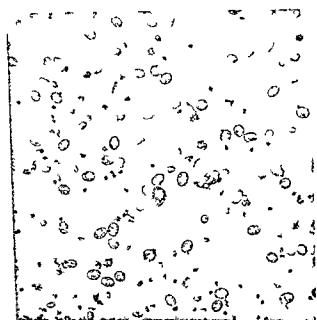
FIG 29 3 – Eimeria sp Fecal smear Approximately X 200

surface epithelium causing destruction and loss of the cells in addition to a mild cellular reaction which results in a slight thickening of the walls

DIAGNOSIS

Coccidiosis is diagnosed by fecal examination (Figs 29 3 and 29 4) A direct smear in water or saline should suffice though concentration by salt or sugar solution will not destroy the oocysts

Species identification is rather difficult and usually not necessary However, Isospora suis can readily be separated by mixing the fecal material with 2 6 per cent potassium dichromate solution and allowing the mixture to stand several days for sporulation to occur (Figs 29 5 and 29 6) Presence of oocysts in the feces should be correlated with symptoms and history

TREATMENT AND CONTROL

The most effective therapeutic agents are the sulfonamides However in most cases, the animals showing clinical signs are past the stage for optimum benefit. Alicata (1946) observed beneficial results from sulfaguanidine at 1 gm per 10 lb of body weight when given two days before infection until 7 days after When fed at the same rate starting after symptoms and fed for three consecutive days no benefit was noted but a reduction in oocyst output was realized Biester and Murray (1933) did not find any therapeutic effect from the use of large repeated doses of colloidal iodine Aureomycin at the level of approximately 50 mg per pound of feed has been reported to be of value (Lederle and Co 1952) Symptomatic treatment of the diarrhea and the prevention of secondary infections is indicated
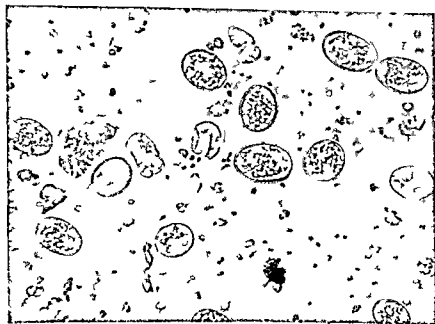
FIG 29 4 – Eimeria sp Fecal smear Approximately X 700

accumulate in large numbers on a single
red blood cell

TRANSMISSION

The natural method of transmission has
not been proven, but, from the nature of
the organism, it is suspected that blood
sucking arthropods play a role The use
of contaminated needles and instruments
must also be included as possible trans
mitting agents In utero transmission as
described by Berrier and Gouge (1954)
may account for its appearance in suckling
pigs

LESIONS AND CLINICAL SIGNS

In clinical cases the disease manifests
itself as an acute, febrile, ictero anemic dis
ease in shoats and is most prevalent in the
summer months The morbidity and mor
tality are low The characteristic course
includes a temperature of 104 to 107° F,
depression, anorexia, and a severe and
rapid blood destruction concurrent with a
drop in the number of parasites Later
signs are icterus, weakness, and the appear
ance of bile stained feces The red blood
cell count may drop to 1 to 2 million

Recovered animals apparently remain car
riers for life The incubation period is
about 6 to 10 days Necropsy findings show
icterus, yellow liver, soft, enlarged spleen,
and thin, watery blood There may be
hydropericardium, ascites, and a pale,
flabby heart in some cases Microscopic
lesions are a hyperplastic bone marrow,
hemosiderosis of the liver, and some ne
crosis of the liver lobules

DIAGNOSIS

Demonstration is based upon the identi
fication of the Eperythrozoon in blood
smears (Fig 29 8) and upon the herd and
individual history

TREATMENT AND CONTROL

Specific therapy against E suis has been
accomplished with two drugs The older
treatment is the use of Neoarsphenamine
as a single intravenous injection at the
rate of 15–45 mg per kg of body weight
(Splitter, 1950c) Similar results have been
obtained using oxytetracycline or tetra
cycline at the rate of 3 mg per pound of
body weight or greater (Splitter and
Castro 1957) Symptomatic treatment,
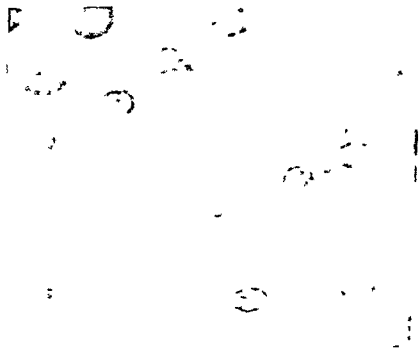
FIG 29 8 — Eperythrozoon su-
is Blood smear Approximate
ly X 1,000

TRANSMISSION

Toxoplasmosis is transmitted in nature by a variety of methods Which of these methods is most important remains to be proven It has been shown to be transmitted in utero through the milk, by ingestion of infected tissues, and, experimentally by arthropods (American dog tick, Rocky Mountain tick, Lone Star tick and the human body louse) The apparent real danger of transmission to humans by the ingestion of improperly prepared or raw pork should be considered

LESIONS AND CLINICAL SIGNS

The acute form is most frequently observed in young pigs and is characterized by signs of respiratory involvement such as coughing and dyspnea Acutely ill pigs have a temperature elevation of 104 to 107° F, shivering, weakness, incoordination, and diarrhea Asymptomatic sows may farrow weak, premature, or stillborn pigs Necropsy reveals fibrinous pneumonia focal necrotic hepatitis and enteritis and lymphadenitis Microscopic examination shows the toxoplasma in the affected areas with the lesions being those of a focal necrosis and granulomatous infiltration The brain exhibits subendymal, focal, and perivascular microglial granulomata and necrosis

DIAGNOSIS

Demonstration of the organism in the tissues by histological methods or animal inoculation is necessary for positive diagnosis The Sabin Feldman dye test and complement fixation tests are of value when a rising titer can be demonstrated In view of the data of Feldman (1953) showing the incidence of antibodies in normal appearing swine and other domestic animals, a single test is of limited value

TREATMENT AND CONTROL

Most treatments have been on an experimental basis, however, two groups of compounds appear promising, (1) the sulfapyrimidines and sulfapyrazines, and (2) the 2 4' diaminopyrimidines, the most active being the pyrimethamine The effective dosage for swine has not been determined for either of these two drugs however, the synergistic action of the two makes it possible to use lower dosages if they are used in combination No control program can be recommended as the exact method of transmission in swine is not known If the dye test or the complement fixation test becomes readily available, the purchase of negative titer pigs for replacement to the herd and the elimination of animals with a titer should reduce the incidence of the disease

REMARKS

The lack of an easy and reliable diagnostic test for toxoplasmosis and the large numbers of asymptomatic cases make this disease a difficult problem The public health hazard is a potential threat when one considers the fact that toxoplasma has remained viable for at least 25 days in body fluids at 4° C

Eperythrozoonosis

This condition has also been described under the names of Icteroanemia and Anaplasmosislike disease There are apparently two species of *Eperythrozoon* in swine, *E suis* (Splitter, 1950a, b) and *E parvum* (Splitter, 1950b). The latter parasite is apparently innocuous, while the former is capable of producing a febrile disease primarily found in the young animal

MORPHOLOGY

E suis, the larger of the two, averages about 0 8μ in diameter but may range up to 2 5μ in some instances The more common shape is the ring form, however, coccus, rod, and budding forms have been described These forms are usually found adherent to the red blood cells, although at times they may be free in the plasma *E parvum* is smaller, averaging about 0 5μ in the ring form This parasite tends to

roncitoi have been described in swine, the former from Russia and Tanganyika and the latter from Italy. These parasites are tick transmitted and resemble the babesia of cattle and dogs in morphology and life history.

Balantidiosis

There is some disagreement as to the validity of species designation of *suis* for the ciliate found in swine. It is this author's opinion that, until further evidence is presented, it should be considered to be *Balantidium coli* (Malmsten 1857.)

MORPHOLOGY

The motile or trophozoite form is oval and varies considerably as to length (30–150μ) and width (25–120μ). The entire body is covered with spiral longitudinal rows of cilia. At the anterior end there is a peristome leading to a cytostome and cytopharynx. The posterior end contains an indistinct cytopyge or excretory pore.

There are two nuclei: a large kidney-shaped macronucleus, and a small spherical micronucleus which lies in the curve of the macronucleus. The non-motile cyst stage is spherical and varies from 45 to 65μ in diameter.

TRANSMISSION

Infections are acquired by ingestion of the cysts or trophozoites as contaminants of feed and/or water.

LESIONS AND CLINICAL SIGNS

Due to the fact that *Balantidium coli* is found throughout the world in both healthy and diseased pigs, it is hard to ascertain the exact lesions and symptoms of this condition. This infection may take place as a secondary condition to some other change such as altered intestinal flora, stress, etc. The infection may be found in both the younger and older animals and is frequently most severe in fat hogs. The initial symptom is a diarrhea
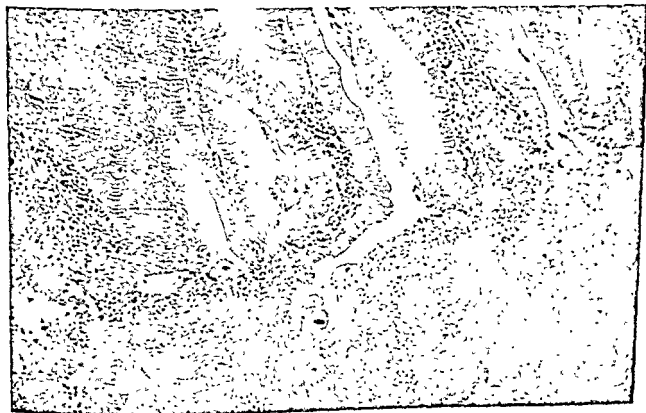
FIG. 29.9 — Balantidium coli. Colon. Approximately X 200.

such as sodium cacodylate, is advantageous for recovery but has no direct effect on the parasite As the exact transmitting agent is unknown, the control measures should be directed against blood sucking arthropods, and care should be taken to insure that instruments used in swine work are sterile

REMARKS

This condition is apparently more prevalent than the literature would indicate the majority of infections being sub clinical in nature Removal of the spleen will frequently result in an increase in the number of organisms found on the erythrocytes The fact that cold weather tends to terminate the spread of cases would indicate that an arthropod vector is involved This is strengthened by the fact that Splitter (1952) found that *E suis* was able to survive for 31 days in frozen blood The finding of a virus associated with cases of anemia as reported by Foote *et al* (1951) does not preclude the fact that there may be two conditions with different etiological agents but similar symptoms and lesions The findings in the two diseases are primarily those of hemolytic anemia irrespective of cause

Sarcosporidiosis

The exact taxonomic status of *Sarcocystis* is uncertain The more recent work seems to indicate that this is a fungus infection rather than protozoan Regardless of the classification, a brief description will be given

MORPHOLOGY

Sarcocystis miescheriana (Kuhn 1865) is found primarily in the striated muscles The form found in the muscle is a cystic stage which varies in size depending upon the location and age of the cyst These cysts will vary from microscopic in size up to the size of a pinhead The cyst, which is called a Miescher s tube, is double walled and contains many spores which have been given the name, Rainey s corpuscles These spores are elongated crescents or spindle shaped The form is not constant and depends on the number formed in the sac

TRANSMISSION

The work of Spindler *et al* (1946) indicates that infection in swine probably takes place by ingestion of feces from animals that have fed on infected flesh

LESIONS AND CLINICAL SIGNS

Most infections are probably unnoticed, however, in heavy infections, the animals have diarrhea, temperature rise weakness in the loin, and posterior paralysis Necropsy findings are pale kidneys, and hyperemia of the stomach and intestinal mucosa The muscles may be watery, light colored and contain small white spots In old cases the cysts may be calcified Microscopically, the cysts are found in the connective tissue between the fibers with little cellular infiltration around the viable cysts

DIAGNOSIS

This condition is diagnosed on necropsy examination and histological examination of the muscles, primarily the diaphragm and heart

TREATMENT AND CONTROL

There is no known treatment for this condition Control measures are rarely necessary, but good nutrition and sanitation to prevent fecal contamination should be practiced

REMARKS

The pathological changes due to *Sarcocystis* may be due to a toxin, rather than to the parasite itself

Other Sporozoan Diseases

Other parasites from the class Sporozoa have been reported from areas other than North America These conditions are not well described, and the exact extent of the diseases is not known Among these infections *Babesia trautmanni* and *B per*

which may be watery and may be intermittent in nature The animal becomes depressed, loses weight and appetite, and may have an elevated temperature Death may occur in 1 to 3 weeks, but usually the animals slowly recover Stunting may occur as a result of the infection in young animals Necropsy reveals extensive colitis In severe cases this colitis may be hemorrhagic The mucosa is swollen and congested on the tips of the folds and may have a necrotic grayish white or yellowish white scab like exudate on the surface Microscopic examination reveals the organism throughout the exudate, deep in the villi, and in the submucosa

DIAGNOSIS

This is accomplished by identification of the characteristic trophozoites or cysts (in large numbers) in the feces or from mucosal scrapings of the colon (Figs 29 9, 29 10, and 29 11) The elimination of viral and microbial pathogens should be attempted

TREATMENT AND CONTROL

There is no specific therapy for this condition Use of nonspecific symptomatic drugs to alleviate the diarrhea and sulfonamides and/or antibiotics to prevent secondary bacterial invasion should be attempted Diets high in carbohydrates favor growth of the organism while high casein diets diminish the number of organisms in the intestine

REMARKS

This parasite is very important from the public health aspect, as in man it causes a serious diarrhea The incidence in humans is confined largely to people having close association with swine The use of hog manure for truck crops should be discouraged unless the material is sterilized Personal hygiene should be promoted among persons having contact with swine

REFERENCES

ALICATA, J E 1916 Occurrence of *Eimeria spinosa* in swine raised in Hawaii Jour Parasit 32 514
———, AND WILLETT, E L 1946 Observations on the prophylactic and curative value of sulfaguanidine in swine coccidiosis Amer Jour Vet Res 7 94
ANDREWS, J S, AND SWINDLE, L A 1952 *Eimeria spinosa* recovered from swine raised in Maryland and Georgia Proc Helm Soc Wash 19 61
AVERY, J L 1942 Survival on soil of oocysts of two species of swine coccidia, *Eimeria debliecki* and *E scabra* Jour Parasit 28 27
BECK, J D, BOUCHER, W B, AND POPPENSIEK, G C 1943 Infectious balantidiosis in swine Jour Amer Vet Med Assn 102 59
BECKER, E R 1934 Coccidia and Coccidiosis of Domesticated, Game, and Laboratory Animals and of Man Iowa State University Press Ames Iowa
BERRIER, H H AND GOUGE, R E 1954 Eperythrozoonosis transmitted in utero from carrier sows to their pigs Jour Amer Vet Med Assn 124 98
BIDERSTEIN, E L, BARR, L M, LARROW, L L, AND ROBERTS, S J 1956 Eperythrozoonosis of swine in New York State Cornell Vet 16 288
BIESTER, H E, AND MURRAY, C 1929 Studies in infectious enteritis of swine IV Intestinal coccidiosis Jour Amer Vet Med Assn 75 705
———, AND ——— 1933 Studies in infectious enteritis of swine VII Studies on the use of colloidal iodine in swine coccidiosis Jour Amer Vet Med Assn 82 79
———, AND ——— 1934a Studies in infectious enteritis of swine VIII *Isospora suis* n sp in swine Jour Amer Vet Med Assn 85 207
———, AND ——— 1934b The occurrence of *Isospora suis* n sp in swine a preliminary note Jour Amer Vet Med Assn 84 291
———, AND SCHWARTE, L H 1932 Studies in infectious enteritis of swine VI Immunity in swine coccidiosis Jour Amer Vet Med Assn 81 358
BRUCE, DAVID, HARVEY, D, HAMERTON, A E, DAVEY, J B, AND BRUCE, M E 1912 The morphology of *Trypanosoma simiae*, sp nov Proc Roy Soc London Series B 85 477
BRUMPT, E J A 1909 Démonstration du rôle pathogène du Balantidium coli Enkystement et conjugation de cet infusoire C R Soc Biol (Paris) 67 103
BURROWS, R B AND KLINK, G K   1955 *Endamoeba polecki* infections in man Amer Jour Hyg 62 156.

FIG. 29.10 — Balantidium coli. Colon. Approximately X 700.

FIG. 29.11 — Balantidium coli. Fecal smear. Approximately X 700.

BUTTREY, B W 1956 A morphological description of a tritrichomonas from the nasal cavity of swine Jour Protozool 3 8

COLE, C R , DOCTON, F L., CHAMBERLAIN, D M , SANGER, V L PRIOR, J A , AND FARRELL, R L. 1953 Toxoplasmosis in domestic animals Proc 15th Int Vet Cong (Stockholm)

—— SANGER, V L FARRELL, R E , AND KORNDER, J D 1954 The present status of toxoplasmosis in veterinary medicine No Amer Vet 35 265

CREECH, G T 1922 Sarcosporidiosis of swine associated with advanced degenerative changes in the musculature Jour Amer Vet Med Assn 61 383

DIAMOND L S., AND RUBIN R 1936 Susceptibility of domestic animals to infection with *Trypanosoma cruzi* from the raccoon Jour P irasit 42 21

DICKE, W E. 1931 Anaplasmosis like disease in swine Vet Med 29 288

DIKMANS G 1915 Check list of the internal and external animal parasites in North America Amer Jour Vet Res 6 211

DIMOCK, W W HEALY, D J., AND HOEFT, G L 1922 Infectious necrotic enteritis in swine No Amer Vet 3 405

—— SNYDER E M., AND HEALY, D J 1922 Infectious necrotic enteritis in swine No Amer Vet 3 339

DOYLE L. P 1932 A rickettsia like or anaplasmosis like disease in swine Jour Amer Vet Med Assn 81 668

EICHENWALD H F 1936 The laboratory diagnosis of toxoplasmosis Ann New York Acad Sci 64 207

EYLES, D E 1956 Newer knowledge of the chemotherapy of toxoplasmosis Ann New York Acad Sci 64 252

FARRELL R L, DOCTON F L., CHAMBERLAIN, D M , AND COLE, C R 1952 Toxoplasmosis I Toxoplasma isolated from swine Amer Jour Vet Res 13 181

FAUST, E. C 1955 Animal Agents and Vectors of Human Disease Lea and Febiger Philadelphia

FELDMAN, H A 1953 The clinical manifestations and laboratory diagnosis of toxoplasmosis Amer Jour Trop Med and Hyg 2 420

FITZGERALD, P R HAMMOND D M., AND SHUPE, J L 1954 Studies on the role of trichomonads in the production of atrophic rhinitis in pigs Cornell Vet 44 302

—— JOHNSON, A E., THORNE, J and HAMMOND, D M 1955 Experimental infection of the bovine genital tract with trichomonads from swine Jour Parasit 41 17

FOOTE, L. E., BRACK, W E., AND GALLAHER, B 1951 Ictero anemia, eperythrozoonosis or ana plasmosis like disease of swine proved to be caused by a filterable virus No Amer Vet 32 17

FRENKEL, J K. 1956 Pathogenesis of toxoplasmosis and of infections with organisms resembling toxoplasma Ann New York Acad Sci 64 215

FRYE W W., AND MELENEY, H E 1932 Investigations of *Endamoeba histolytica* and other intestinal protozoa in Tennessee. IV A study of flies rats, and mice and some domestic animals as possible carriers of the intestinal protozoa of man in a rural community Amer Jour Hyg 16 729

—— AND —— 1934 Studies of *Endamoeba histolytica* and other intestinal protozoa in Tennessee. VIII Observations on the intestinal protozoa of young pigs and attempts to produce infection with a human strain *E histolytica* Amer Jour Hyg 20 404

GOLDSTEIN, H E 1953 Progress of atrophic rhinitis studies Vet Med 48 223

HAMMOND, D M., AND FITZGERALD, P R 1953 Observations on trichomonads of the digestive tract and nose of pigs Jour Parasit 39 11

—— AND LYND, N 1957 Experimental infections of the genital tract of swine and goats with *Trichomonas foetus* and *Trichomonas* species from the cecum or feces of swine Amer Jour Vet Res 18 461

HENRY D P 1931 A study of the species of *Eimeria* occurring in swine Univ Calif Publ Zool 36 115

JACOBS L 1956 Propagation, morphology, and biology of toxoplasma Ann New York Acad Sci. 64 151

KINSLEY, A T 1933 Amebic enteritis in swine Vet Med 25 312

KOFOID, C. A., AND DONAT, F 1933 Experimental infection with *Trypanosoma cruzi* from the intestine of the cone nosed bug, *Triatoma protracta* Proc. Soc. Exper Biol and Med 30 489

KUDO, R R 1927 Pathogenic protozoa of domestic animals Jour Amer Vet Med Assn 71 466

—— 1954 Protozoology Charles C Thomas Springfield, Ill

LAWLESS, D K 1954 Report on a human case of *Endamoeba polecki* Jour Parasit. 40 221

LEDERLE AND COMPANY 1952 Aureomycin Lederle Laboratories Division, New York

LEVINE, N D 1940 The effects of food intake upon the dimensions of *Balantidium coli* from swine in culture Amer Jour Hyg 32 81

—— MARQUARDT, W C., AND BEAMER, P D.. 1954 Failure of bacteria free trichomonas to cause atrophic rhinitis in young pigs Jour Amer Vet Med Assn 125 61

MERCK AND COMPANY 1955. The Merck Veterinary Manual Merck and Co., Rahway, N J

MOMBERG-JORGENSON, H D 1956 Toxoplasmosis of swine Nord Vet Med. 8 227

SECTION V

TOXEMIAS AND POISONINGS

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CHAPTER 30

Coal-Tar Poisoning and Mercury Poisoning

Coal-Tar Poisoning

Coal tar poisoning is an acute and often fatal disease Its clinical course usually progresses without the appearance of noticeable physical symptoms, death often being the first sign of illness Lesions of the liver are perhaps the most important indication of the disease Clay pigeon poisoning and pitch poisoning are other names used to designate this disease

ETIOLOGY

Poisonous substances in coal tar pitch are the primary etiological factor The specific active principle or principles in coal tar pitch that are responsible for the poisoning have not been identified, therefore, the cause is ascribed to the coal tar pitch mixture or compound as a whole

Quin and Shoeman (1933) described a disease of the liver in swine as an idiopathic hemorrhagic hepatitis for which a cause had not been found, although subsequently they implicated clay pigeons Graham et al (1940) were the first to discover that this degenerative liver disease resulted from the ingestion of fragments of expended clay pigeons

After correlating the ingestion of clay pigeon fragments with the occurrence of the disease, Graham et al undertook to prove the toxicity of some of the suspected material that was obtained from a farm

where pigs had died from coal tar pitch poisoning A group of five 9 week old pigs were fed a diet compounded from corn oats wheat middlings, tankage, minerals and cod liver oil to which was added a measured quantity of powdered clay pigeons The test substance was fed at the rate of 15 grams per pig per day On the fourth day of the trial the pigs refused the feed mixture Each then was given 6 grams of the powered clay pigeons in a gelatin capsule for another two days All five pigs died 8 to 20 days later At necropsy, four showed evidence of liver injury, but no noticeable lesions were observed in the remaining pig This placed the burden of cause on the clay pigeons Since they were prepared from a mixture of finely powdered limestone and coal tar pitch, the next move was directed to a study of the pitch

A liquid coal tar preparation was put in gelatin capsules and administered to young pigs Three grams were given to each of three pigs for five successive days and all died within 10 to 18 days Pronounced diffuse degenerative changes in the liver were found at necropsy Thus, these workers concluded that the coal tar pitch in clay pigeons was toxic for swine if consumed for a period of several days in daily amounts of approximately 15 grams

Since that time, Giffee (1915), Fenster macher et al (1915), and Aitken (1956)

visible mucous membranes are icteric The mucous membranes of the mouth and eyes are discolored by the bile pigments in the circulating blood The respiratory rate may be increased and show a thumpy type of breathing Death comes to a high proportion of the pigs that manifest symptoms

PATHOLOGICAL CHANGES

The outstanding lesion observed at necropsy in pigs poisoned with coal tar pitch is the altered appearance of the liver When the abdominal cavity of a pig with a typical and fully developed case of coal tar pitch poisoning is opened, the greatly enlarged liver with a variegated mottling

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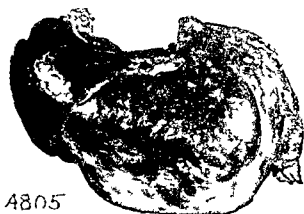
FIG 30 2 — Liver from pig shown in foreground of Figure 30 1 The pig died after 43 days of being fed ground clay pigeons Note the engorgement, the mottled appearance, and the roughened hepatic surface The mottling in some cases is much more extensive than shown here (Photograph by H W Dunne)

makes a striking pathological picture It is engorged and is quite friable The lobular architecture of the liver is very distinct Some of the lobules are dark red in color and others are yellow with a shading toward a copper colored tint The intensity of the color varies between these extremes in other affected lobules This accounts for the characteristic variegated mottling that stands out so sharply When the liver is sliced, the mottling shows up distinctly on the cut surfaces An excess of fluid in the peritoneal cavity is not uncommon The lymph nodes of the abdominal cavity are swollen and hemorrhagic As a rule, the kidneys are enlarged and turgid and somewhat pale in color No other significant lesions have been found in the other organs or tissues The subcutaneous tissues and mucous membranes are frequently yellowish or orange in color indicative of a jaundiced condition The necrosis of the liver cells and vascular tissues of the lobules allows bile to enter the circulation Its subsequent distribution throughout its body produces jaundice

From the standpoint of microscopic changes the lobules are either partially or almost completely filled with blood Generally the hemorrhage begins at the center of the lobule and extends toward the periphery, but sometimes it occurs only in the midzonal portion The red cells in some lobules show evidence of destruction and lysis with the presence of hemosiderin Other lobules show the changes characteristic of a central necrosis in which the cells of the liver cords are swollen and have a very granular cytoplasm with small and densely stained nuclei Some cells may have undergone autolysis and appear as an amorphous substance

TREATMENT

There is no known treatment for this disease When its presence in a herd is recognized it is advisable to determine the source of the offending compound and to take the necessary steps to prevent the pigs from coming in contact with it

It is important to know that a pasture can be contaminated with coal tar pitch for long periods of time The history on one of our cases revealed that approximately 35 years prior to the time of the occurrence of the coal tar pitch poisoning, an area of the pasture where the losses were occurring had been used as a target range for shooting clay pigeons More often however, the history indicates that it is a period of a year or two since the contamination occurred

have reported losses in swine from coal tar pitch poisoning

Other sources of coal tar pitch considered to be responsible for fatal cases of this liver disease have been reported Giffee (1945) described cases that appeared to have been due to the consumption of tar that was used for sealing and surfacing a pipeline for the transportation of gas The history of a case examined by the writer suggests a similar source In this instance the affected pigs had ' chewed off ' and consumed the tar substance on lumber dismantled from a tank that had been used for storing water The pigs had access to the tar from this source for two or three weeks prior to their sudden death In another of our cases the cause of death was traced to a tarry sludge that contaminated a small area of a pasture lot occupied by the affected pigs The sludge or residue came from an establishment engaged in cleaning and re storing steel drum containers which had been collected from many different sources and which had been used for various pur poses A spill-over from a drainage ditch that carried wastes from this establishment had flooded the area of the pasture and left shallow pools of oily water that slowly seeped away leaving a tarry sludge or resi due on the surface of the soil The source was not detected until a careful inspection of the pasture was undertaken None of the

pigs in adjoining but uncontaminated lots was affected by this disease

Another interesting case, reported by Fenstermacher et al (1945), shows the necessity of continuing the search for a likely source of coal tar pitch The history disclosed that the pig had developed a habit of eating tarred roofing paper which had been placed around the base of several farm buildings as a protection against low temperatures and frost However, our ex perience and the experience of others with whom we have communicated indicate that clay pigeons are the source of the toxic com pound in most outbreaks

CLINICAL SIGNS

The sudden death and rapid clinical course of this disease often occur without the appearance of symptoms of diagnostic significance Under these circumstances death is the only physical sign indicative of the existence of a morbid process How ever, some animals live for several hours or even days after the clinical onset, in which case the affected pigs usually show signs of physical weakness and depression They are recumbent much of the time and gener ally lie in the sternal position The res piration rate is increased and a tenderness over the abdomen can be detected by digi tal palpation The disease is afebrile A secondary anemia usually develops and the
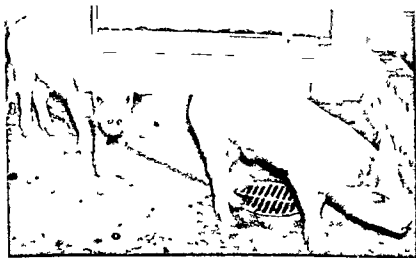
FIG 30 1 — Two pigs which had been fed small amounts (5 grams) of ground clay pi geons in their feed daily for 35 days Note the nasal dis charge, lassitude, and loss of flesh (Photograph by H W Dunne)

PATHOLOGICAL CHANGES

Gross tissue changes in subacute mercury poisoning are not striking and are of little to no diagnostic value In some of the more chronic cases that came to our notice the kidneys were enlarged and very firm in consistency They were also very pale On histological section, they showed an extensive interstitial fibrosis and tubular degeneration McEntee (1950) records a coagulation necrosis of the convoluted tubules and also of neurons in the brain The colon in some of the induced cases showed a pronounced diphtheritic inflammatory exudate on the surface of the mucosa the latter was also hemorrhagic

DIAGNOSIS

On the basis of present information and knowledge of mercury poisoning in swine, it is not possible to cite one or more clinical or pathological features which would differentiate it from other poisons or poisonous substances However, by carefully and thoroughly assembling case histories, it will be disclosed in many instances that some of the grain the pigs were consuming had been treated with a mercurial fungicide This should be strong circumstantial evidence for the existence of mercury poisoning The ultimate diagnosis depends upon demonstrating the presence of mercury in the tissues, especially of the kidneys and liver, from the affected porcine carcass In the specimens collected from experimentally induced cases, the least amount found was 17 mg of mercury per 100 grams of kidney tissue (Ferrin et al, 1919)

TREATMENT

Nothing definite or specific is known with respect to the treatment of swine poisoned by mercury On the other hand if circumstances are such that an effort to treat seems desirable it is suggested that the pigs be given milk to drink in place of water They may also be given two raw eggs per 60 lb of body weight every 8 hours for 3 days Deep intramuscular injections of BAL (British anti lewisite) may be beneficial since the drug has the property of inactivating mercury that is already absorbed (Sollmann 1918) It is dispensed as a 10 per cent solution in oil and administered at the rate of 1 mg per pound of body weight and repeated in one to two hours It may be necessary to give a third and a fourth injection but inasmuch as BAL is toxic in large amounts, the third and later doses are reduced to 1 mg per 2 lbs of body weight and the interval between injections lengthened to 12 hours

Poisoning from mercury should not be allowed to occur in swine Seed grains that have been treated with any mercury containing fungicide must not be fed to swine nor to other livestock It is recommended that any surplus of treated grain be burned and the ash buried deep in the earth

REFERENCES

Coal-Tar Poisoning

AITKEN, W A 1956 Coal tar poisoning in pigs Jour Amer Vet Med Assn 128 262
FENSTERMACHER, R , POMEROY, B S AND KERNKAMP, H C H 1945 Pitch poisoning in swine
    Proc 49th Ann Meet U S Livestock San Assn, p 86
GIFFEE, J W 1915 Clay pigeon (coal tar) poisoning in swine Vet Med 40 97
GRAHAM R , HESTER, H R , AND HENDERSON, J A 1940 Coal tar pitch poisoning in p 56 J of
    Amer Vet Med Assn 96 155
QUIN, A H , AND SHOEMAN, J D 1953 Idiopathic hemorrhagic hepatitis Jour Amer Vet Med
    Assn 82 707

Mercury Poisoning

FERRIN, E F, KERNKAMP, H C H, ROLLAF, M H, AND MOORE, M B, 1949 Treated seed grain
    found fatal to hogs Minn Farm and Home Science 6 7
McENTEE, K 1950 Mercurial poisoning in swine Cornell Vet 40 143
SOLLMANN, T 1918 Pharmacology 2th ed W B Saunders Philadelphia and London p 875
TAYLOR, E L 1917 Mercury poisoning in swine Jour Amer Vet Med Assn 111 66

Mercury Poisoning

Poisoning from mercury is not of un common occurrence Reports of it have not been numerous, but we have reason to believe that many cases occur which are not properly diagnosed It occurs most often in a subacute form but acute cases also are recognized For the most part the poisoning results from the ingestion of seed grains treated with fungicides that contain mercury For this reason many cases occur in the summer season or several weeks after completion of the seeding operations on the farm Gastrointestinal, renal, and nerv ous disturbances are usually manifested by swine suffering from mercury poisoning Treatment has not been satisfactory

ETIOLOGY

Swine are most frequently exposed to mercury in the form of organic and inor ganic mercurial compounds The active principles of many fungicides employed in the control of fungous diseases of oats, wheat, barley, and flax are organic mer curial compounds Both dry and liquid preparations containing approximately 2 to 3 per cent mercury equivalent are available for this purpose, and then only a small amount of the fungicide is mixed with the grain Taylor (1947) and McEntee (1950) report the loss of swine from consuming grains that had been treated with mercurial fungicides The treated grains were fed for 6 to 12 weeks before signs of disorder were manifested The time interval for the occurrence of symptoms depends somewhat upon the concentration and amount of the injurious substance ingested

In a controlled study of the harmful ef fects from the ingestion of treated seed grains Ferrin et al (1949) fed to a group of pigs, oats that had been treated with a fungicide containing the equivalent of ap proximately 2 per cent of mercury The fungicidal preparation was mixed with the grain according to the recommended pro cedure which was ½ oz. to 1 bushel of grain It was fed ad libitum After about 20 days of feeding, symptoms of illness were ob

served, followed by death in another 5 to 10 days Those that received the treated grain for only 10 days showed no signs of any disorder due to the mercury, and they eventually reached market weights Like wise, the control pigs that consumed un treated oats remained healthy at all times

The chloride of mercury, or calomel, is a source of mercury that, at times, may be the cause of mercury poisoning Such poi soning is usually the result of "over dosing based upon the questionable premise that ' if a little is good, more is better'

CLINICAL SIGNS

The clinical course of mercury poisoning, when physical symptoms are manifested, is usually fairly rapid As a rule, swine poi soned by mercury die in 5 to 10 days after the onset of symptoms The course, how ever, bears a direct relation to the amount of the poisonous substance consumed The symptoms are not pathognomonic for poi soning by mercury, but they are sufficiently distinctive to suggest that the cause of the disorder is of the nature of a poison or a poisonous substance This is especially true of the symptoms present several or more hours prior to death

Anorexia is usually the first physical symptom to be noticed This is soon fol lowed by signs of general physical weak ness When standing, the pig tends to sway from side to side and moves off with an un steady gait McEntee (1950) reports a glos sopharyngeal paralysis as an early sign A disturbance of vision is manifested by er ratic attempts to move about Later the pig becomes prostrate and produces a paddling motion with its feet The pig is semi comatose in this stage and has no fever Death from uremia usually results from extensive renal damage

Vomiting, diarrhea, and colicky pains occur in the acute form of mercury poison ing Also a very marked physical weakness and prostration is noted in the acute cases and death appears to be due to a circula tory collapse

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CHAPTER 31

Sodium Salt Poisoning

For more than 100 years, salt poisoning in swine has been an enigma The first account in the English scientific literature of the condition appeared in 1856, it de scribed a report by a German veterinarian, M Adam In the same year, Lepper (1856) wrote that as far back as 1816 he had ob served the phenomenon associated by Adam with the feeding of brine At that time the symptoms had been attributed to the 'stagger bone' said to be located in the palatine portion of the mouth Attempts were made by quacks of the day to cure the condition by removing a portion of bone from this area

A great many clinical reports of poison ing by brine or salted foods appear in the literature Among the more graphic de scriptions of the condition are those by Pyatt (1862), Junginger (1887), Lamour eux (1890), Parker and Brooksbank (1949), Wautie (1935), and Kernkamp (1919)

ETIOLOGY

Sodium chloride poisoning occurs more frequently in swine than in any other do mestic animal Pigs may be more suscep tible to this form of poisoning, but the facts that they are often fed garbage or other food of unknown composition are kept in inadequately equipped premises and are victims of all sorts of careless hus bandry may be reasons for the higher in cidence of the disease in this species

Field outbreaks of salt poisoning have been associated with the ingestion of rock salt, pickling brine, salted fish the fallout from heavily salted hays, and excess sodium chloride in buttermilk, whey garbage, and commercial feeds One outbreak was due to kitchen waste containing powdered soap in which the offending agent was sodium carbonate Insufficient trough space which can cause pigs to eat quickly and greedily and the provision of salt in quantity after a period of salt starvation are other fac tors that have resulted in the ingestion of toxic doses Diets deficient in certain nutri ents such as vitamin E and sulphur con taining amino acids may increase the susceptibility of pigs to poisoning (Hjarre and Obel, 1956) Experimentally, the dis ease has been produced by feeding sodium chloride (Bohstedt and Grummer, 1954, Smith, 1954, Hjarre and Obel, 1956), sodium propionate (Smith, 1955a), pow dered soap containing sodium carbonate (Moore, 1898) and sodium lactate (Hjarre and Obel, 1956)

Unless the dose of salt is very large the effect of the salt is relative to the amount of water that is consumed by the pig, for water apparently acts as a vehicle for eliminating excess sodium chloride via the kidney and bowel For this reason, re striction of water intake, feeding without providing a separate supply of water, and hot weather, which increases the loss of fluid from the body, can influence the

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and death within two days after the salt is consumed

Acute sodium salt poisoning more particularly concerns us here since it is the form that usually results from the voluntary consumption of sodium salt by pigs and is therefore frequently met with in the field It is a clear cut, readily recognizable, pathological and clinical entity Pigs that have been given a high salt, low fluid diet show thirst, pruritus, and constipation After 1 to 5 days some of them will appear blind and deaf, be oblivious to their surroundings, show no interest in food or drink, and will not respond to external stimuli Affected pigs will wander around aimlessly, bumping into and pushing against objects on reaching a corner, a pig may attempt to continue walking and force its snout up the wall Occasionally pigs exhibit pleurothotonus and pivot around one front or hind foot Forced circling is commonly seen At this stage of the disease the pig may recover, or become comatose and die within a few hours, or develop epileptiform seizures

In the majority of animals, epileptiform seizures occur (Fig 31 1) with remarkable regularity at 7 minute intervals The onset of an attack is signalled by twitching of the snout This is followed in sequence by clonus of the neck muscles and circling or running movements Occasionally contractions of the cervical muscles result in stepwise upward movements of the head which will jerk to an almost perpendicular position, causing the center of gravity to shift involuntarily toward the hind quarters Compensatory efforts to maintain normal posture may cause the pig to move rapidly backwards like a horse backing a heavy load (Fig 31 2), or it may assume a sitting position with the nose pointing upwards (Fig 31 1) A seizure lasts up to 1 minute and, in typical cases, ends in profuse salivation, rigidity, respiratory arrest and cyanosis After an attack the pig may collapse and remain in coma for a variable period of time or get up and wander aimlessly until the onset of another attack It may die during a seizure on the other hand, the attacks may suddenly cease to occur and, after a short period of readjustment during which it will regain its sight and take an interest in its surroundings, the pig will appear quite normal

As a result of exertion during an attack pulse and respiration rates increase and temperature rises, but these rapidly return to normal However, in cases occurring in hot weather, temperatures up to 108° F have been recorded, and death from heat stroke can occur unless preventive measures are instituted

The characteristic signs have commenced as early as 39 hours, and death has occurred as early as 47 hours after a pig was placed on a high salt, low fluid diet Some pigs have not shown signs of poisoning until the sixth day of salt feeding and a few have recovered spontaneously after exhibiting signs for periods up to 7 days (Smith, 1957) The mortality ranges from 0 to 100 per cent, with an average in 20 reported outbreaks of 3 per cent.

FIG 31 1 — Epileptiform seizure typical of acute salt poisoning (Selected frame from 16-mm Kodachrome motion picture film)

PATHOLOGICAL CHANGES

Clinical pathology. Increases in blood serum sodium levels are consistently present during the period of high salt feeding When sodium chloride is the offending agent, the chloride serum levels are also significantly increased However, by the time signs of poisoning appear, the pig has

toxicity of a particular amount of salt Sodium salt poisoning can be produced in pigs simply by adding pure sodium chloride to their regular ration and limiting, for a time the supply of water It can occur in pigs receiving as little as 2 5 per cent sodium chloride if water is given only at intervals and the amount restricted On the other hand, when a continuous ample supply of fresh water is available poisoning may not occur in pigs receiving a ration containing 10 to 13 per cent sodium chloride A 20 per cent aqueous solution of sodium chloride given by stomach tube to 4 month-old pigs at a level higher than 2 2 gm per kg body weight has caused peracute poisoning (Smith, 1955b)

Following a time on high salt, low fluid diet, there appears to be a critical period during which the ingestion of a large amount of water favors the development of salt poisoning On one experiment 1 8 gm per kg body weight produced the classical signs and lesions of acute sodium salt poisoning In this case each pig was restricted to 2 liters of water on the first day after receiving the salt and 2 liters on the second and was then given 5 liters on the third (Smith, 1957) In field outbreaks and in experimental trials, pigs which appeared unaffected when the herds were first observed, developed signs of poisoning 16 to 24 hours after the salted feed was removed and they were given free access to water (Hjarre and Obel, 1956 Smith 1955b) Failure to recognize this critical relationship between the salt consumed by the pig and the amount of fluid taken at that time or the amount taken later probably accounts for unsuccessful attempts to produce the disease (Worden, 1941), the great variation in estimation of the toxic dose (Glasser et al, 1950, Volker, 1950), and the opinion formerly held by some that a toxic factor other than salt was responsible for the syndrome in swine

PATHOGENESIS

The pathogenesis of sodium salt poisoning is not fully understood, but present knowledge indicates that the sodium ion

and a body fluid volume disturbance are complementary factors It has been proposed (Smith, 1955b, 1957) on the basis of known facts that if an excess of salt is ingested and water intake is not increased immediately, a high blood sodium level will be reached and, by a process of impeded diffusion, an increased concentration of sodium in the brain will result The blood sodium level will subsequently decrease, becoming especially low if a large amount of water is taken by the pig at this stage The osmotic gradient thus created may result in edema of the brain and in increased intracranial pressure, hence reduction of the blood supply Utter (1950) has proven that sodium is a strong inhibitor in the brain of anaerobic glycolysis through stimulation of conversion of adenosine triphosphate to adenosine monophosphate (AMP) and a decreased rate of removal of AMP by phosphorylation The resulting accumulation of AMP inhibits glycolysis The combination of reduced oxygen supply resulting from the high intracranial pressure and the inhibition of anaerobic glycolysis may cause degeneration of the specialized tissue of the cerebral cortex The clinical signs and the lesions in the brain, except the selective attraction of the eosinophils, may be explained on this basis

CLINICAL SIGNS

There are two forms of sodium salt poisoning in pigs — peracute and acute, the peracute occurs when a massive dose has been administered, and the acute form when lesser amounts of sodium salts are consumed over a period of time and water intake is restricted A third syndrome called chronic salt poisoning ' and characterized by dehydration, stiffness and anorexia, has been described, but investigation of the literature and of suspected cases usually suggests that chronic salt poisoning is due to lack of water rather than to excessive sodium salt intake

The signs of peracute sodium salt poisoning are great weakness, muscular tremors, running movements, prostration, coma,

FIG 31 3 — D stent on of
pericellular spaces with
d ffuse and focal vacuola
tion indicating edema n
the inner zone of the cere
bral cortex X 120

FIG 31 4 — Many eosino
ph ls around blood ves
sels in cerebrum X 120
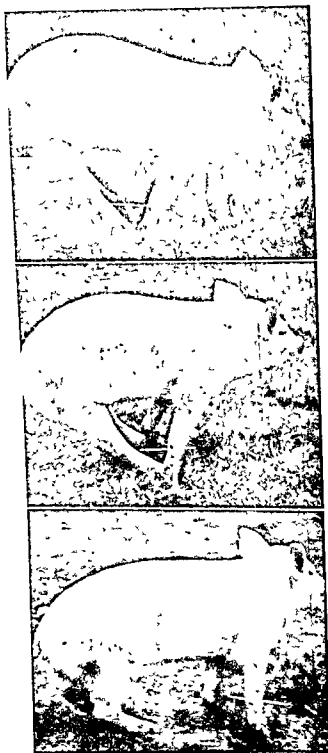
FIG. 31 2 — A characteristic maneuver likened to a horse backing a heavy load Seen during seizure in acute salt poisoning. (Selected frames from 16 mm. Kodachrome motion picture film)

ceased to eat and within 24 hours the level of sodium in the serum may fall to only slightly above normal. Eosinophils disappear from the blood stream at the onset of the signs. This probably represents a response to the severe stress and therefore is not specific. A subsequent increase in these cells signals recovery.

Necropsy. Occasionally the mucous membrane of the stomach and intestines is inflamed, but they may be normal in appearance. Ulcers occur in the gastric mucous membrane of pigs that have experienced many epileptiform seizures. If the animal has died shortly after the onset of signs, congestion and edema of the meninges and the cerebral cortex will be observed.

The microscopic changes occurring in the central nervous system are considered to be pathognomonic. These consist of a meningoencephalitis characterized by edema (Fig. 31.3) and infiltration of eosinophils into the overlying meninges and around the blood vessels of the cerebral cortex (Fig. 31.4). These alterations may be seen most advantageously in pigs that die shortly after the onset of symptoms. In very early cases the eosinophils will be few in number, and in some sections are found only in the meninges in the depths of the sulci. Polioencephalomalacia with proliferation of vascular endothelium and glial cells becomes evident later (Fig. 31 5). Cystic spaces may be formed as the softened tissue is removed by the scavenger cells derived from the microglia (Fig. 31.6). Eosinophils are gradually replaced by round cells. The lesions are seen most frequently in the cerebral cortex, principally in that part of the cortex which forms the walls of the inner third of the sulci.

DIAGNOSIS

In typical cases of sodium salt poisoning, diagnosis can be made on the basis of clinical signs alone. The clinical picture is similar to that in enterotoxemia (gut edema), but the altered squeal and response to external stimuli, typical of enterotoxemia, are absent.

Sodium salt poisoning may be diagnosed at necropsy on the basis of the microscopic changes in the cerebral cortex In acute cases the classical eosinophilic meningo encephalitis and malacia will be observed Increase in the blood serum sodium level is another significant diagnostic feature Chemical analyses of the stomach contents may reveal excess sodium salt, a negative result, however, can be misleading since sodium is rapidly absorbed from the gastro intestinal tract

TREATMENT

There is no specific treatment for salt poisoning The efficacy of any treatment is difficult to assess since a dramatic spon taneous recovery is frequently observed Calcium therapy has been suggested Wautie (1935) gave limited data to indi cate that the blood calcium level is low in pigs with salt poisoning and cited clinical experience to show the value of calcium gluconate therapy as a specific treatment. But more recent studies carried out on pigs affected with acute salt poisoning have shown that the calcium level in the blood serum is normal In one trial, a solution containing dextrose, calcium, and mag nesium appeared to be effective in one pig, which abruptly ceased having seizures and made a dramatic recovery, however, others treated in the same way failed to respond (Smith, 1955b)

Chloral hydrate has been suggested as a specific treatment by Bormann et al (1885), who reported favorable results from its use in pigs poisoned by herring brine

When salt poisoning occurs in a herd, the feed should be replaced immediately pending an examination to determine its sodium salt content However, a normal sodium level in the feed may be misleading since signs of illness usually do not appear in the pigs until several days after initial exposure to excess salt, by which time all the salty feed may have been consumed

Water intake should be strictly con trolled The giving of a large amount of water at this critical stage may favor the development of the disease in these animals Fresh water in small amounts at first may be given to those pigs which are not show ing signs In experiments the majority of observably affected swine that were not given fluids recovered spontaneously after several days They were kept in a cool place and given adequate space and ample bedding to prevent injury during con vulsions (Smith 1955b) This suggests that pigs showing signs of salt poisoning should not be forced to take water since it does not appear to be necessary and under certain conditions, may even be harmful Drenching must be avoided because the semiconscious pigs are also predisposed to inhalation pneumonia

PREVENTION

The amount of sodium salt in the diet of pigs should be carefully controlled A liberal supply of fresh water, other than that given with the feed, should be avail able to pigs at all times This is particu larly important if garbage or other ration of unknown composition is being fed Ob servations on experimental animals indi cate that pigs will not voluntarily consume sufficient sodium salt to cause poisoning if they are given adequate trough space and water is available in quantity

REFERENCES

Anonymous 1856 On the poisonous properties of brine Veterinarian 29 366
Bohstedt, G , and Grummer R H 1954 Salt poisoning of pigs Jour Anim Sci 13 933
Bormann Stern, and Schafer 1885 Vergiftung durch Heringslake und Heilung durch Chloral hydrat Arch wiss prakt Tierheilk 11 25,5
Glasser K , Hutka E and Wetzel, R 1930 Die Krankheiten des Schweines 9th ed M and H Schafer, Hannover p 335
Hjarre A , and Obel A L 1936 Kochsalzvergiftung als Ursache einer Meningo Encephalitis eosinophilica beim Schwein Monatsh f Vet Med 11 690
Junginger, E 1887 Kochsalzvergiftung bei Schweinen Wschr f Tierheilk Viehzucht 31 137
Kirkbride, H C H 1919 Salt poisoning in swine Cornell Vet 9 58
Lamoureux M 1890 Vingt six porcs empoisonnés par la saumure Rec med vet 7 511

FIG 31 5 — Dystrophic cal cification with prolifera tion of endothelial and glial cells indicating heal ing in an area of necrosis in the cerebral cortex X 120
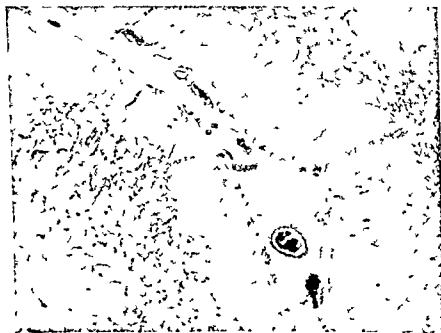
FIG 31 6 — Malacia with cyst formation All zones of the cortex except the outer layer are involved The space is populated with scavenger cells and bridged by capillaries X 40

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CHAPTER 32

Toxic Plants, Rodenticides, Herbicides, Lead, and Yellow Fat Disease

TOXIC PLANTS

There are many toxic plants but a rather small number of them cause poisoning in swine Plants which are especially poison ous in the early growth stages or plants which contain the toxic principle in the roots are most often the cause of such poisonings Most of the other toxic plants poisonous to swine are rarely consumed by them due to their feeding habits and the types of rations fed them

Cocklebur

Xanthium pennsylvanicum and other species These plants are widely distributed throughout the world They may be found any place but most cases of poisoning oc cur when the plants are growing along fences banks of streams or ditches on over flowed land or the beds of dry ponds Poisoning of pigs results from their con suming the seedlings Eating of the spiny burs may cause mechanical irritation of the intestine and the burs may mat to gether to cause obstruction

The seeds and young plants while in the cotyledon stage contain the toxic glucoside xanthostrumarin The amount of xantho strumarin decreases as the leaves develop

CLINICAL SIGNS

Within a few hours after a pig ingests a toxic amount of cocklebur seedlings the

following symptoms develop depression nausea vomition weakness ataxia sub normal temperature rapid pulse and respi rations Spasms of the neck muscles may occur Death may occur within a few hours after symptoms appear

PATHOLOGICAL CHANGES

Hyperemic areas in the mucosa of the stomach and intestine may be found due to the irritant effect of xanthostrumarin Petechiae may be found in the cortex of the kidney and on the myocardium

TREATMENT

Oral administration of mineral or raw linseed oil may delay absorption of the xanthostrumarin Intramuscular injection of 5–30 mg physostigmine may produce dramatic response in some cases Dosage is for pigs from 30 to 250 lb It may be repeated in 30–60 minutes Physostigmine will contract skeletal muscle and aid in recovery from muscle weakness characteris tic of this poisoning It slows the heart and increases intestinal motility due to its para sympathomimetic effect which is pro duced by inactivation of choline esterase

Nightshade

The leaves and green berries contain several alkaloids the most abundant of which is the glucoside solanine The soil

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LETTER H 1856 On poisoning of pigs with brine Veterinarian 29 434

MOORE V A 1898 Powdered soap as a cause of death among swill fed hogs Jour Comp Med and Vet Arch 19 306

PARKER, W H AND BROOKSBANK, N H 1949 Suspected salt poisoning in pigs Vet Rec 61 f

PLATT, H 1862 Poisoning of pigs with common salt Veterinarian 35 768

SMITH D L T 1954 Salt poisoning in swine Rep New York State Vet Coll 1952 1953, p 30

——— 1955a Salt poisoning in swine Proc Book, Amer Vet Med Assn, 92nd Ann Meet p 69

——— 1955b Sodium salt poisoning in swine Ph D thesis, Cornell Univ, Ithaca, N Y

——— 1957 Poisoning by sodium salt — a cause of eosinophilic meningoencephalitis in swine Amer Jour Vet Res 69 825

UTTER M F 1950 Mechanism of inhibition of anaerobic glycolysis of brain by sodium ions Jour Biol Chem 185 499

VOLKER, R 1950 In Eugen Frohner Lehrbuch der Toxikologie fur Tierarzte, 6th ed Ferdinand Enke, Stuttgart p 54

WAUTIE, M 1935 Contribution à l'étude des accidents causés par le sel et la saumure chez le porc Ann Méd Vét 80 253

WORDEN, A N 1941 Salt poisoning in pigs Vet Rec. 53 695

of five alkaloids conine, methyl conine conhydrine, pseudo conhydrine, and coniene They are found in all parts — roots stem, leaves, and seed The root is said to contain only a small amount of the toxic principle in the spring

CLINICAL SIGNS

The action is very rapid There is a brief period of salivation followed by trembling of the flank muscles, depressed respirations and rapid weak pulse, muscle weakness and paralysis The body temperature may rise several degrees There is paralysis of the peripheral endings of the motor nerves and there is evidence that the drug also has an effect on the sensory nerves Hayashi and Muto (1901) have shown the phrenic nerve to be more susceptible to the conium alkaloids than are the other motor nerves of the body Death from poison hemlock is due to respiratory failure

PATHOLOGICAL CHANGES

This poisoning does not result in any typical tissue changes

TREATMENT

The stomach should be emptied if possible Physostigmine (5–30 mg) should be administered subcutaneously and repeated in 30 minutes if necessary Artificial respiration is indicated

St -John's-wort

This plant is widespread in North America and many other countries The poisonous principle is a volatile oil and two fluorescent substances, hypericin and hypericum red Eating of this plant when in the flowering stage leads to photosensitization and subsequent dermatitis of the unpigmented portions of the skin upon exposure to the sunlight. Animals usually do not eat the plant if other feed is available Dermatitis does not develop if animals are not permitted in the sunlight

CLINICAL SIGNS

There is elevation of body temperature, often up to 105° F The pulse is rapid as

are respirations Some animals may have diarrhea White animals develop a dermatitis on exposure to sunlight Blisters form and often necrosis of the skin and subcutaneous tissues occurs

PATHOLOGICAL CHANGES

Inflammation in the stomach and intestine in addition to the dermatitis and possible necrosis of white areas of the skin are the characteristic lesions

TREATMENT

The animals should be placed where they will not be exposed to the sunlight and a soothing antiseptic ointment applied to the external lesions Any ointment containing a lanolin or petrolatum base and some anti infective agent is satisfactory A ration composed of finely ground grain and protein supplement should be provided

Buttercup

There are several members of the Ranunculus family and all contain the toxic active principle anemonol This toxic principle is an acrid, volatile constituent strong enough to produce blisters if placed in contact with the skin The members of this family are usually found growing in pastures or woodlands where there is a good supply of moisture *Ranunculus acris* (tall buttercup) is very common in pastures and meadows and is probably the cause of most cases of poisoning by buttercups The dried plants seldom contain sufficient active principle to cause poisoning

CLINICAL SIGNS

There is evidence of abdominal pain and most hogs develop diarrhea There is twitching of the muscles of the ears, nervousness, dyspnea tachycardia, and paralysis Pigs may be in a paralyzed state for 2 or 3 days before death

PATHOLOGICAL CHANGES

Inflammation of the stomach and intestine is usually severe Petechiae may be distributed over the mucosa, although

on which the plant is grown, climate, and maturity of the plant influence the amount of toxic principle present As the berries ripen, the toxic agent gradually decreases to nontoxic amounts There are many plants in the nightshade family capable of causing toxicity in pigs The common potato, *Solanum tuberosum*, contains sol anine and solanidine in the 'eyes' and new sprouts The black nightshade *So lanum nigrum*, which is widely distributed in the United States is the common cause of this type of poisoning

CLINICAL SIGNS

Poisoned animals show signs of stupe faction loss of appetite, constipation, mus cular trembling incoordination, convul sions and coma Dilation of the pupils and rapid pulse and respirations are also noted Some poisoned pigs vomit The body temperature usually remains normal

PATHOLOGICAL CHANGES

Pigs which die suddenly from night shade poisoning may show no lesions If the animals live for some time after symp toms of poisoning develop, extensive in filtration of tissues around the kidneys with blood tinged serum may occur In mature hogs large blood clots may be found adjacent to the kidney

DIAGNOSIS

The toxic principle of this plant is rapidly eliminated by the kidneys Urine from an animal suspected of being poi soned by nightshade may be instilled into the eye of a test animal (cat, rabbit dog) to determine whether it will dilate the pupil

TREATMENT

Parasympathetic stimulant drugs are in dicated One to 5 mg of carbachol (len tin), 5–30 mg of physostigmine, or 10–50 mg of pilocarpine may be given by sub cutaneous or intramuscular injection and repeated at 30–60 minute intervals if neces sary

Water Hemlock

Water hemlock is an erect branched leafy herb with a purple streaked stem up to 6 feet tall The roots are large and form tubers which may be 3 to 4 inches long The tubers, which appear similar to pars nips or artichokes, contain the resin like cicutoxin, the toxic principle The tubers of which only a small amount may fatally poison an animal, are toxic all seasons of the year

CLINICAL SIGNS

The following signs are observed in poi soned swine frothing at the mouth, nerv ousness nausea, vomition, weak and rapid pulse, dyspnea, and intermittent convul sions The body temperature increases and may be 106° F after a convulsion In fatal poisoning the convulsions become more violent Death results from respiratory failure

PATHOLOGICAL CHANGES

No specific changes due to the toxic agent have been reported

TREATMENT

An emetic such as 50 ml of 1 per cent cupric sulfate, or apomorphine at the rate of 0 02 mg per pound of body weight followed by 30–120 gm of sodium sulfate may save the animal if a fatal amount of the toxic principle has not been absorbed Convulsions may be controlled by giving pentobarbital sodium at the rate of 7 mg per pound of body weight, or chloral hydrate 1 ml 6 per cent solution per pound of body weight These solutions should be injected intravenously or intra peritoneally

Poison Hemlock

This coarse biennial herb has a smooth purple spotted stem and leaves that re semble parsley The leaves, when bruised smell like parsnips The root is long and resembles a parsnip in appearance The toxicity of this plant is due to the presence

the poison Oxygen should be administered, but no fluids should be given

Sodium Fluoroacetate

This white crystalline, odorless, tasteless water soluble chemical has been found toxic to all animals The pig is one of the domestic animals most susceptible to the toxic action of this compound, the fatal dose being 0 3 mg per kg of body weight (McGirr and Papworth, 1955) Animals which eat rodents poisoned by this compound may get a fatal dose

It produces its effect by two mechanisms (1) stimulation of the central nervous system and (2) alteration of cardiac function which results in cardiac depression arrhythmias, and ventricular fibrillation

CLINICAL SIGNS

Nausea and vomition may appear in pigs 30 to 60 minutes after ingestion of sodium fluoroacetate There is an initial period of nervousness and tetanic muscular spasms The repeated spasms lead to exhaustion followed by toxic depression of respiratory and vasomotor centers The tissues become cyanotic Pulse rate is very rapid and weak, cardiac arrhythmias and general circulatory depression rapidly appear There is an initial rise in body temperature, but it falls and may be subnormal in severely poisoned animals

The course is rapid, death may occur within 30 minutes to several hours after symptoms appear Few animals that develop marked symptoms recover

PATHOLOGICAL CHANGES

There are numerous subepicardial hemorrhages on a heart which has stopped in diastole The blood is very dark red and tarry in appearance The tissues are very dark red in color The spleen and liver are congested and swollen

TREATMENT

To induce vomiting, 50 ml of 1 per cent cupric sulfate solution should be administered orally

Intravenous administration of pentobarbital sodium (15 mg per pound of body weight) will overcome the stimulant effect of sodium fluoroacetate on the nervous system Repeated doses may be necessary Glyceryl monoacetate (monoacetin) administered intramuscularly at 0 2 ml per pound of body weight every half hour for six hours has been used to control cardiac fibrillation in laboratory animals poisoned with sodium fluoroacetate (Gleason et al, 1957) There are no reports on the use of this drug in poisonings in pigs

Red Squill

Preparations of squill employed as rat poisons are derived from the dried bulbs of the sea onion Urginea maritima a plant indigenous to Mediterranean countries The active principles are glycosides some of which have a digitalis like action on the heart Squill has the reputation of being a safe rodenticide based on its unpalatability to domestic livestock and the fact that usually, when consumed it is vomited Palatability trials show that under normal farm feeding conditions it is very unlikely that pigs would voluntarily eat sufficient red squill bait to be poisoned (Fitzpatrick, 1952)

CLINICAL SIGNS

In pigs poisoned with red squill there is hyperesthesia, depression, weakness, ataxia, cardiac arrhythmias, extrasystoles dyspnea cyanosis, and paralysis Vomiting may or may not occur The course of poisoning seldom exceeds 3 days Death occurs as a result of cardiac arrest

Symptoms usually develop within 6 hours after ingestion Diarrhea may develop in mature swine but it is seldom observed in young pigs

PATHOLOGICAL CHANGES

Gastritis and enteritis are usually marked with congestion, edema hemorrhage, and often ulceration of the mucosa There is congestion of the mesenteric vessels, and the mesenteric glands are swollen,

hemorrhage into the lumen of the intestine is uncommon The lungs are congested and petechiae are found on the periphery The body temperature shows an initial rise, but as paralysis develops it drops to subnormal

TREATMENT

Mineral oil should be administered to act as a protective to the alimentary tract It also will act as an intestinal lubricant to hasten elimination of the unabsorbed toxic principle, anemonal Supportive treatment, especially administration of glucose, is indicated to maintain the animal, if possible until the effect of the toxic principle subsides Twenty per cent glucose solution (250–1,500 ml) may be injected intraperitoneally Saline solution or a combination of glucose and saline solution may be injected intraperitoneally to maintain the fluid balance

RODENTICIDES

Some rodenticides have the reputation of being safe because they are either unpalatable to domestic animals (red squill) or repellent to man and domestic animals because of odor (zinc phosphide) There are, however, several rodenticides in use which are readily ingested by, and are toxic to, domestic animals

Antu

Alpha naphthyl thiourea (Antu) is a white crystalline powder (most commercial preparations have a blue-gray tint) which is highly insoluble in water, is stable to heat and which deteriorates very little in dry storage It has no perceptible odor and only a transient bitter taste There is variation in susceptibility of some species to the toxic action of Antu, depending upon age There is, however, no evidence indicating variability of toxic effects on pigs of different ages The amount of ingesta in the stomach does appear to influence the potential danger from toxicity of this compound because an animal with an empty stomach usually vomits as a result of the irritant action of the chemical on the gastric mucosa and therefore may not be poisoned

The course of Antu poisoning is rapid most animals die within a few hours after ingesting the compound A single dose of 40 mg per kg of body weight may be fatal repeated consumption of sub toxic doses does not produce symptoms or lesions (McGirr and Papworth, 1955)

CLINICAL SIGNS

It kills by its action on the capillaries of the lungs, producing pulmonary edema Respiratory symptoms characteristic of pulmonary edema — inspiratory and expiratory dyspnea, dullness on percussion, and moist rales on auscultation of the thorax — develop rapidly Coughing may occur The pulse is rapid and heart sounds are not distinct Visible tissues become cyanotic The temperature becomes subnormal Diarrhea may develop late in the course of the poisoning The animal becomes comatose and dies from hypoxia induced by the drowning pulmonary edema

PATHOLOGICAL CHANGES

Edema of the lungs with hydrothorax, hyperemia of the tracheal mucosa acute gastroenteritis, hyperemia of the kidneys, and pale liver are constant changes observed

DIAGNOSIS

Chemical examination of tissues and fluids may confirm diagnosis The extraction procedure described by Jones et al (1949) gives reliable results if death has occurred or specimens are obtained within 24 hours after ingestion of Antu

TREATMENT

Effective antidotes are not available Prompt emptying of the stomach by administering an emetic (50 ml of 1 per cent cupric sulfate solution, or apomorphine at the rate of 0 02 mg per pound of body weight) may reduce further absorption of

FIG. 32.1 — Pig fatally poisoned with warfarin on day before death. Note the bleeding at the nose and the blood on the sides of its head. The tongue is protruded and the mouth is open in an attempt to facilitate breathing. (Photograph by H. W. Dunne.)
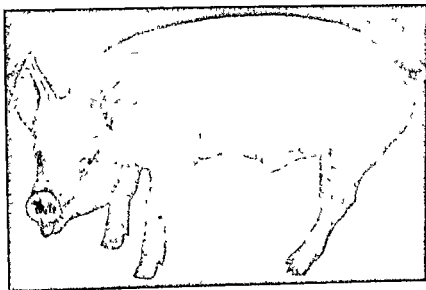
there may be several liters of blood in the peritoneal cavity.

TREATMENT

Transfusion of blood from a normal healthy pig to the affected pig is the most effective treatment. Vitamin K may be administered to stimulate prothrombin production. An emulsion of 5 per cent vitamin K (phytonadione) in 5 per cent dextrose solution, given intravenously, 50 mg. per 100 lb. of body weight, will quickly reverse the hypoprothrombinemia (Link, 1957). Intramuscular injection of a similar dose of vitamin K is effective but not as rapid in action as intravenous injection.

HERBICIDES
Dinitro Compounds: Dinitrophenols, Dinitrocresols

These compounds, which are yellow crystalline materials, are highly toxic to swine. They are readily absorbed through the skin and lungs. Poisonings can occur if animals are sprayed accidentally or have access to herbage that has been recently sprayed. Residues that remain on foliage which has been treated for some time are not dangerous to animals.

CLINICAL SIGNS

These compounds cause an extraordinary increase in the oxidative processes of the body. There is a rapid elevation of body temperature up to 106° F., tachycardia, dyspnea, nervousness, convulsions, coma, and death. Exposure to dinitro compounds usually causes a yellowing of the skin around the mouth or on other parts of the body where there may have been contact.

PATHOLOGICAL CHANGES

Yellowish discoloration of the skin around the mouth and of the buccal mucosa is almost always observed. There is acute swelling of the liver and kidneys. The mucosa of the stomach and intestine may be yellow depending upon the interval since ingestion of the toxic agent.

TREATMENT

No specific antidote is available. The stomach should be washed with a 5 per cent solution of sodium bicarbonate, leaving some of the solution in the stomach. An intravenous injection of 5 per cent dextrose and physiological saline solution (250–1,000 ml.) should be given, followed by a stimulant such as caffeine and sodium benzoate (0.5–3 gm.) or camphorated oil (20 per cent, 2–5 ml.) intramuscularly.

Plant Hormones

2,4-D, 2,4,5-T, and esters of these compounds have been tested for toxicity. Data for the fatal oral dose for swine are not available. Acute oral toxicity studies in the

edematous, and congested kidneys, liver lungs and myocardium are congested and swollen There may be areas of necrosis in the liver and degeneration of the cells in the kidney tubules

TREATMENT

The animal should be isolated, and undue exertion which might further strain the impaired circulation should be prevented Emetics or gastric lavage may be used if the interval since ingestion of the squill indicates that some may remain in the stomach

Thallium Sulfate

This chemical is used infrequently as a rodenticide It has proved to be toxic to all species to which it has been administered

CLINICAL SIGNS

The effect of thallium is rapid in onset It has some local emetic properties and pigs may vomit after ingesting it Salivation, evidence of abdominal pain, diarrhea dyspnea, tachycardia weakness, impaired vision, hyperesthesia, convulsions, and coma are observed in this type of poisoning Animals which recover from thallium poisoning are often blind and may lose most of their hair In chronic poisoning there is loss of hair, reddening of the skin and moist eczema about the eyes and mouth in addition to the other symptoms described

PATHOLOGICAL CHANGES

The mucosa of the tongue and mouth may be hyperemic if death is sudden There may be ulcerative stomatitis if the animal lives for some time after poisoning There is ulcerative gastritis and enteritis and degeneration of the kidney tubules

TREATMENT

Sodium or magnesium sulfate (25–125 gm) should be given to produce catharsis Demulcents such as milk or bismuth subnitrate may be given Intravenous administration of 20–40 ml of 1 per cent solution of sodium iodide followed by 40–60 ml of 10 per cent sodium thiosulfate to eliminate

the thallium is relatively effective Injections of calcium gluconate or other calcium preparation and a parasympathetic stimulant such as pilocarpine (10–50 mg) antagonize the action of thallium

Warfarin

Warfarin (3 α phenyl β acetylethyl 4-hydroxy coumarin) is a tasteless, odorless chemical which inhibits the formation of prothrombin and causes capillary hemorrhage

It is closely related chemically to dicoumarin which has been isolated from moldy sweet clover hay It is potentially dangerous to all classes of animals and birds The lethal dose of Warfarin for swine has not been determined, but this species is very susceptible to its toxic properties (McGirr and Papworth, 1955) Poisonings have occurred from accidental contamination of feed (Reihart and Reihart, 1952), consumption of treated meal or malicious use of the chemical

CLINICAL SIGNS

Evidences of Warfarin toxicity are slow in onset It does not destroy vitamin K, but evidence suggests it competes with it in enzyme systems essential for prothrombin production Although it inhibits formation of prothrombin in the liver almost immediately after ingestion, the prothrombin present in the plasma must be exhausted before evidence of poisoning appears This requires 24 to 48 hours depending upon the age and condition of the animal Lameness, anemia, swellings on the legs or parts of the body that may have been bruised, and rapid respiration and heart beat are apparent in most pigs poisoned with Warfarin Occasionally there is some bleeding from body openings

PATHOLOGICAL CHANGES

Examination of the blood reveals a low erythrocyte count and prolonged clotting time There is usually some free blood in the body cavities Hemorrhages may be found in almost any tissue of the body, particularly the lungs In mature hogs

tion in the intestine is hemorrhagic in character and shreds of mucous membrane may be eliminated The animals become dehydrated although they usually drink large quantities of water There is marked distress due to the abdominal pain The body temperature may be elevated 3 to 4 degrees Affected animals may be down and unable to rise Chronic poisoning causes symptoms similar to those of acute poisoning, but the onset is more insidious and therefore may be misleading

PATHOLOGICAL CHANGES

The gastrointestinal tract is edematous and hemorrhagic The mucous membrane may have sloughed from areas of the intestine, and in severe poisoning there may be perforation of the stomach or intestinal wall There are areas of focal necrosis in the liver and degeneration of tubule cells in the kidneys Some tubules may be blocked with coagulated protein The spleen is congested and enlarged The lungs are also congested There are petechiae under the endocardium and ecchymosis on the left ventricle

DIAGNOSIS

Chemical examination of liver or kidney tissue, or stomach contents obtained at necropsy, may confirm diagnosis Examination of urine by the Reinsch test may aid in diagnosis

TREATMENT

If time since exposure has been short enough to indicate that arsenic may still be in the alimentary tract, treatment should be directed toward removal of the material by administration of an antidote, lavage, or a cathartic A protein material such as egg white may serve as a temporary antidote Since the arsenic protein combination is a rather loose one, it must be promptly removed to be of much value Lavage is the best procedure for such removal Dimercaprol (Bal) is the treatment of choice Two mg per pound of body weight injected intramuscularly is the recommended dosage This dosage

should be repeated at 4 hour intervals the first 24 hours and daily thereafter until recovery Intravenous injection of 10–20 ml of a 25 per cent solution of sodium thiosulfate is considered of value if administered before marked symptoms appear

LEAD

Paint containing lead discarded batteries and contaminated vegetation containers or water are the main sources of lead poisoning

CLINICAL SIGNS

Symptoms develop rapidly in young pigs but may be slow in onset in mature hogs There is loss of appetite and evidence of gastroenteritis with passage of grayish white feces The feces may become very dark gray and be tinged with blood There is salivation champing the jaws frenzy blindness convulsions coma and death Mature animals usually have diarrhea and show incoordination especially in the hind legs, blindness, prostration, and death

PATHOLOGICAL CHANGES

Hemorrhagic gastroenteritis is a fairly constant finding The liver may be pale and degenerated with areas of necrosis Renal hyperemia with hemorrhage and subepicardial and subendocardial hemorrhage are commonly observed Examination of blood cells reveals stippling of the erythrocytes

DIAGNOSIS

Blood from an affected animal may be analyzed for lead content by the method recommended by Hammond et al (1956) Tissue (liver) obtained at necropsy may also be analyzed by the same method to confirm clinical diagnosis

TREATMENT

Treatment of lead poisoning consists in avoiding the source of lead and in removing the lead from the digestive tract by giving magnesium sulfate (25–125 gm) in aqueous solution Protein in the form of

dog indicate the LD_{50} is 100 mg per kg of body weight The dosage necessary to produce toxicity in laboratory animals is greater than that for the dog

Results of observations made when swine were pastured in areas freshly treated with 2 4 D or 2,4,5 T indicate that there is no hazard of toxicity involved (Grigsby and Farwell, 1950) The only possible danger results from an accumulation of nitrites in certain weeds such as pigweeds and ragweeds which have been sprayed with these compounds (see Nitrates)

As a group these chemicals have been shown to be nontoxic to experimental and farm animals under practical conditions When large doses have been administered experimentally, depression with loss of appetite, accompanied by loss of weight muscular weakness and incoordination have been noted

Nitrates

Nitrates are not as toxic as are nitrites The nitrates are not as readily converted to nitrites in the alimentary tract of swine as in ruminants Definite information on the minimum toxic dose of nitrate for swine is not available A common source of nitrate poisoning in swine is the nitrate fertilizers There is a possibility that forage which has been sprayed with a herbicide may contain an excess amount of nitrate but a number of factors, such as amount of nitrogen in the soil, available moisture, and temperature, appear to influence the change in nitrate content Immature barley, pigweed, and frosted beet tops are possible plant sources of nitrates Nitrate poisoning from water supplies is due to pollution of the water by drainage from barnyards or other places of manure concentration or drainage from fields recently treated with nitrate fertilizers Some water supplies naturally contain abnormal amounts of nitrates or nitrites

CLINICAL SIGNS

The initial symptoms are those of gastroenteritis due to the irritant effect of the chemical There is an increase in respiratory rate, and the pulse becomes rapid and weak Salivation, dilation of the pupils, polyuria and opisthotonos are characteristic of this type of poisoning Weakness ataxia, and cyanosis develop due to conversion of hemoglobin to methemoglobin by the nitrite ion resulting in tissue anoxia and to the low blood pressure resulting from action of nitrite on the blood vessels

PATHOLOGICAL CHANGES

The blood is a chocolate brown color due to the methemoglobin Gastric and intestinal mucosa are often hemorrhagic In some animals there are erosions in the gastric mucosa Petechiae may be found on serous surfaces Areas of emphysema are found in the lungs The liver and kidneys are hyperemic but have color similar to that of the blood The heart and lungs may also be chocolate brown in color

TREATMENT

Intravenous injection of 4 mg of methylene blue per pound of body weight in a 4 per cent solution to convert the methemoglobin to hemoglobin is recommended (Link, 1957) Oral administration of mineral oil or other protective for the gastric and intestinal mucosa is indicated

Arsenic

Some herbicides contain arsenic although there are other sources of arsenic to which swine may have access Several insecticides contain arsenic and are a common source of arsenic poisoning

CLINICAL SIGNS

Acute poisoning usually is observed a few hours after exposure The first signs are nervousness, muscle twitching, tremors, nasal discharge, diarrhea, and weakness The pulse is feeble and fast Inflammation of the stomach and intestine is followed by edema, then by rupture of the blood vessels and necrosis of epithelial and subepithelial tissues There may be perforation of the stomach or intestine The profuse diarrhea which results from the inflamma

GORHAM, J R, BAE, N, AND BAKER, G A 1951 Experimental yellow fat disease in pigs Cornell Vet 41 332

GRIGSBY, B H, AND FARWELL, E D 1950 Some effects of herbicides on pasture and on grazing livestock Mich Agr Exp Sta Quart Bull 32 378

HAMMOND, P B, AND SORENSEN, D K 1957 Recent observations and treatment of lead poisoning Jour Amer Vet Med Assn 130 23

———, WRIGHT, H N, AND ROEPKE M H 1956 A method for the detection of lead in bovine blood and liver Minn Agr Exp Sta Tech Bull 221

HAYASHI, M, AND MUTO, H 1901 Action of conium alkaloids on nerves Arch Exper Path 48 96

HILDITCH, T P 1947 The Chemical Constitution of Natural Fats 2nd ed John Wiley and Sons New York

HOLM, L W, RHODE, E A, WHEAT, J D AND FIRCH G 1953 Treatment of acute lead poisoning in calves with calcium disodium ethylenediaminetetraacetate Jour Amer Vet Med Assn 123 528

JONES, L M 1957 Veterinary Pharmacology and Therapeutics 2nd ed Iowa State College Press, Ames Iowa

———, SMITH, D A AND SMITH, H A 1919 Antu poisoning in dogs Amer Jour Vet Res 10 160

LINK, R P 1957 Personal observation

McGIRR, J L, AND PAPWORTH, D S 1955 Toxicity of rodenticides Vet Rec 67 124

MARSH, C D 1924 Cockleburs (species of Xanthium) as poisonous plants U S D A Bull 1274

MASON, K E, DAM, H, AND GRANADOS H 1946 Histological changes in adipose tissue of rats fed vitamin E deficient diet high in cod liver oil Anat Rec 94 265

MERCK VETERINARY MANUAL 1955 Merck and Co Rahway N J

MUENSCHER, W C 1941 Poisonous Plants of the United States The Macmillan Co New York

OSTERTAG, R V 1934 Text book of Meat Inspection 1st ed Alexander Eger Chicago

PRIBICEVIC, S, AND SEVKOVIC, N 1954 Poisoning of pigs by Xanthium saccharatum Acta Vet Belgrade 4 58

REINHART, O F AND REINART, H W 1952 Accidental warfarin poisoning of young pigs Vet Med 47 372

ROWE, V K, AND HYMAS, T A 1954 Summary of toxicological information on 2 4 D and 2 4 5 T type herbicides and an evaluation of the hazards to livestock associated with their use Amer Jour Vet Res 14 622

TEHON, L R MORRILL, C C AND GRAHAM R 1946 Illinois plants poisonous to livestock Ill Agr Exp Sta Circ 599

TOURTELLOTTE W W, AND COON J M 1951 Poisonings with sodium fluoroacetate Jour Pharmacol 101 82

milk or egg white may be administered orally to precipitate the lead as an albuminate Tannic acid (5–15 gm) may also be administered to induce the precipitation of lead tannate Edathamil has been used to accelerate the elimination of lead in acutely or chronically poisoned calves (Holm et al, 1953) They employed a dosage of 33 mg per pound of body weight repeated daily for 3 to 7 days Hammond and Sorensen (1957) administered 100 mg per pound of body weight per day divided into two doses and continued for 10 to 14 days Edathamil can be injected intravenously, subcutaneously or intraperitoneally The use of this drug in swine has not been reported but it appears that it should act as a chelating agent in this species Administration of 20 per cent calcium borogluconate (50 ml per 100 lb of body weight) favors deposition of lead in the bones where it is not acutely toxic (Merck Veterinary Manual, 1955)

YELLOW FAT DISEASE

For many years inspectors have noted swine carcasses that have yellow or grayish yellow adipose tissue (Gorham et al, 1951) Investigators have reported that feeding pigs fish fat, fish scraps, or refuse from fish canneries resulted in yellow adipose tissue and a fishy odor in the flesh Present knowledge indicates that two nutritional factors — (1) excessive amount of highly unsaturated glycerides and (2) inadequate amounts of tocopherols — are necessary for the acid fast pigment to accumulate in the adipose tissue (Mason et al, 1946) Approximately 80 per cent of the fatty acids in the body fat of halibut and salmon are of the unsaturated type (Hilditch, 1947)

CLINICAL SIGNS

Pigs fed a ration which contains a large amount of fish scrap develop a rough hair coat, lassitude, weakness, and pale mucous membranes Most affected pigs have a poor appetite and do not gain well and some show occasional lameness A catarrhal ocular exudate is common Some pigs fed a ration which contains a large amount of fish scraps die suddenly The cause of death in such cases has not been explained, but it is presumed to be due to the presence of something in the fish scrap There is hypochromic anemia

PATHOLOGICAL CHANGES

The body fat has a lemon yellow color the skeletal and cardiac musculature are a pale red color and friable Abdominal lymph nodes are swollen, edematous, and may have some small scattered hemorrhages The liver has a tan color, indicating marked fatty changes The kidneys are also a pale red, and on cross section the medulla has a greenish tint The mucous membranes of the stomach and intestine are hyperemic The erythrocyte count is in the normal range, but severely affected animals have low hemoglobin levels

TREATMENT

Removal from the ration of the unsaturated fatty acids of fish origin plus the feeding of 500–700 mg per day of tocopherols should permit correction of the condition Considerable time will be required for removal of all of the acid fast pigment from the tissue

REFERENCES

ANONYMOUS 1951 Unusual case of hemlock poisoning in swine Calif Vet 5 26
BRADLEY, W B EPPSON, H F AND BEATH, O A 1939 Methylene blue as an antidote for poisoning by oat hay and other plants containing nitrates Jour Amer Vet Med Assn 96 11
FITZHATRICK R J 1952 Toxicity of red squill raticide to domesticated animals Jour Comp Path and Therap 62 23
——— McGIRR J L AND PAPWORTH, D S 1955 The toxicity of rodenticides II Red squill and zinc phosphide. Vet Rec. 67 142
GLEASON, M N GOSSELIN, R E., AND HODGE, H C. 1957 Clinical Toxicology of Commercial Products Williams and Wilkins Co Baltimore

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CHAPTER 33

Botulism

Botulism is a highly fatal disease caused by the toxins of *Clostridium botulinum* (or *Cl parabotulinum*) and characterized by a rapidly progressive motor paralysis It is not a bacterial infection, but rather an intoxication caused by ingestion of the pre formed toxins, usually in decomposed or "spoiled" animal or vegetable matter As a disease of man, it was first ascribed to the eating of blood sausage Dolman (1957) states that ' the poisonous tenden cies of blood sausage have been recognized in Europe for over 1,000 years, judging by ancient edicts warning against their con sumption The peculiar form of neuro paralysis liable to follow eating such sausages was first accurately described in Wurttemberg in 1735, while the actual term *botulismus*, or sausage poisoning, ap peared in the medical literature of south ern Germany about 150 years ago in 1820 the poet physician Justinus Kerner published a treatise on botulism, giving details of the symptomatology and inci dence of the disease, and asserting that in such disasters the noxious agent had de veloped within the sausage ." The na ture of this noxious causative agent was first demonstrated by Van Ermengen (1897)

As a disease of domestic animals, it has been recognized since 1917 when Graham et al recognized it in horses, and Dickson and Buckley and Shippen pointed out the striking similarity between botulism in man, ' limber neck in chickens and for age poisoning ' in horses Largely through the work of Graham and his associates, it became evident that it least some of the outbreaks diagnosed as ' forage poisoning" in horses actually were due to the toxins of *Cl botulinum* Of course, many others undoubtedly were encephalitis of viral origin In 1920 Hart described an out break in a large flock of chickens and many such outbreaks of 'limber neck ' or botulism, have since been observed Botu lism in other species seems to be relatively infrequent This is due in part, at least to resistance to the toxin (Dack and Gib bard, 1926a), but perhaps in part also to other factors such as feeding habits For example, there is evidence that certain outbreaks in cattle have been caused by feeding on carrion as a result of perversion of appetite due to mineral deficiency (Theiler, 1927, Schmidt, 1930, Bennetts and Hall, 1938 Sutherland, 1955) In any event, cattle, sheep, swine, dogs, and cats are commonly regarded as relatively re sistant to botulism Wild ducks are suscep tible and large numbers have died from the disease The disease has been reported in captive mink (Hall and Stiles, 1935). The common laboratory animals are sus ceptible, guinea pigs and mice being the animals of choice with which to work experimentally. Current thinking regard ing botulism as a naturally occurring dis ease in swine, as summed up by Scheibner

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evidence that exotoxin is liberated when the bacteria undergo lysis Whether the apparent slight absorption of toxin from the intestinal tract of the pig is related to its presence chiefly in colloidal rather than soluble form remains to be demonstrated

As early as 1920 21, Dickson and Shevry observed that in cats, dogs, and rabbits botulinum toxin did not impair the upper or lower neurons of the skeletal motor nerve supply nor affect the spinal reflex arcs of the extremities They concluded that the nerve impulses were blocked in the nerves of the nonsympathetic portion of the involuntary nervous system and that the blocking was of a temporary nature not due to organic destruction of nerve elements Guyton and MacDonald (1947) later concluded that the toxin probably has no primary action on either muscle or nerve trunk, but causes increased delay of action potential at the myoneural junction and proximal to the acetylcholine producing organ, probably either in the proximal portion of the end plate or in the terminal fibrils

Naturally and artificially affected animals appear to be affected alike After the latent, or incubation, period of 8 to 72 hours, a progressive weakness of the voluntary muscles appears, usually beginning in the head and neck regions and spreading backward over the body The weakness leading to paralysis is manifested in disturbance of several functions Most obvious, of course, is the disturbance of loco-

motion Weakness in the forelegs often appears first, followed by involvement of the hind legs and ultimately prostration due to complete paralysis of all limbs The muscles of deglutition are often affected resulting in inability to swallow and in salivation The ears may droop more than usual adding to the appearance of depression Vision may be impaired Superficial reflexes appear to be affected only in that the motor responses become progressively weaker The activity of smooth muscle does not appear to be affected The chief effects upon the circulatory system are arrythmia and tachycardia (Quin 1916) Ultimate involvement of the respiratory muscles results in cyanosis and other evidences of anoxia and finally death by asphyxia An interesting observation on the terminal asphyxia was made by Hamre (1941) After she gave 100 mouse MLD s of partially purified botulinum toxin to chicken embryos on the 11th 16th and 18th days of incubation significant increase in mortality did not occur until the 20th day of incubation the time when pulmonary respiration is being established This suggests that chicken embryos as well as other animals, probably die of respiratory failure The few cases of botulism which survive may require weeks or even months for complete restoration

PATHOLOGICAL CHANGES

Descriptions of the finer lesions of botulism in swine are apparently nonexistent. Sometimes described as a lesionless disease, it is probable that, on the basis of its economic importance no one has felt justified in investigating it carefully Sisak and Meniowitsch (1929) reported that experimentally produced botulism in guinea pigs rabbits and one dog was characterized by destruction of Nisl granules in ganglion cells of the anterior horn (ventral horn of the spinal cord), medulla and pons accompanied by neuronophagia Shav (1916) reported that the toxin has a destructive action on the hepatic cells, particularly in the centrolobular locations He described vacuolization, rupture of the

FIG 33 1—Hog with botulism Note evidence of weakness of voluntary muscles. (Photo courtesy Dept of Veterinary Pathology and Hygiene, Univ of Illinois)

(1955), is that it is of no great importance, Sutherland (1955) regards it as occurring very rarely if at all Hagan and Bruner (1957) concur

Cl botulinum is widely distributed in nature being found in soils especially those well fertilized, and in fruits, vegetables insects, carrion, hog manure, aquatic and emergent vegetation, and moldy hay (Dickson, 1916 Quortrup and Holt 1941 Cook, 1945)

ETIOLOGY

The organism responsible for the production of the toxins causing botulism is generally referred to as *Cl botulinum* (*Bacillus botulinus*) On the basis of its proteolytic abilities, it is now commonly subdivided into two groups and designated as either *Cl botulinum* (non proteolytic) or *Cl parabotulinum* (proteolytic) However, for purposes of general reference they will be referred to collectively herein under the term *Cl botulinum*

Cl botulinum is a motile rod shaped organism, generally $0 6-1\mu$ wide and $4-8\mu$ long It is Gram positive, particularly when young, and occurs singly or in short chains. Spores are generally terminal or subterminal Growth requires rather strictly anaerobic conditions Liver serves as an excellent enrichment factor Deep agar colonies are fluffy in appearance Glucose, levulose, and maltose are fermented with the formation of acid and gas while the reactions in other carbohydrate media are rather variable Gelatin is rather rapidly liquefied As would be anticipated, the effects of growth in protein rich media such as litmus milk, serum, or egg albumen are variable, those strains designated as *Cl parabotulinum* producing change by their proteolytic activities Growth occurs within a wide range of temperatures up to body temperature, but is perhaps optimal at about 30° C (Hagan and Bruner, 1957, Kelser and Schoening, 1948, Merchant and Packer, 1956)

PATHOGENESIS AND CLINICAL SIGNS

As indicated above botulism is fundamentally an intoxication rather than an infection This is in spite of the fact that massive doses of toxin free spores may rarely result in sufficient germination, multiplication, and toxin liberation *in vivo* to produce the disease (Coleman and Meyer, 1922, Starin and Dack, 1925) Several types of botulinum toxin (A B C, D, and E) have been described as well as some sub types (C_a and C_b) Variation in cultural features does not appear to be too closely related to type of toxin produced, e g, type B toxin may be formed by proteolytic or non proteolytic strains while among either proteolytic or non proteolytic strains more than one type of toxin may be formed

Even though the toxin is said to resist a degree of acidity equivalent to that of the gastric juice and not to be changed in toxicity by the action of pepsin or trypsin (Bronfenbrenner and Schlesinger, 1924), Dack and Gibbard (1926a) found pigs particularly resistant to oral doses of the toxin This may be due to the fact that there is only slight absorption to toxin through the small intestine of the pig (Dack and Gibbard, 1926b) Also there is a possibility that the bacterial flora of the pig s intestine has a deleterious effect on the toxin (Sherman *et al*, 1927)

Swab and Herbert (1933) concluded that the toxin itself is a general protoplasmic poison which has a selective action on peripheral nerves and striated muscle tissues especially the former It is elaborated within the bacterial cell in intimate association with the bacterial globulin Nelson (1927) believed it to be released into the surrounding medium by death and disintegration of the cell, thus, in this sense, it would not be considered a true secretion He found that it could be separated from the globulin by peptic digestion and concluded that it is not identical with that substance Boroff *et al* (1952), obtained type D toxin in both colloidal and soluble forms They were able to split the soluble form (exotoxin) off from the colloidal particles by the action of ultrasonic waves in the presence of catalase They believed that the colloidal form is the precursor of the soluble toxin and offered additional

occur in the same fashion as in contagious diseases *Clostridium botulinum* may oc cur in swine feces but there is no evidence that ingestion of small numbers of the bacilli alone will cause the disease under natural conditions However, swine could serve as a reservoir of contamination (Burov *et al*, 1937).

Production of the pre formed toxin ap parently depends upon getting the right organism in the right medium and under the right conditions of temperature, an aerobiosis, etc For example, milk and other dairy products seldom act as dis seminating agents, probably because some of the bacteria commonly found in milk such as *Streptococcus lactis* and *Lactobacil lus casei*, are capable of destroying the toxin (Sherman *et al*, 1928).

Spoiled canned goods, spoiled garbage, and carrion appear to be the more com monly incriminated media in which botu linum toxin is formed Thus in spite of the fact that swine are relatively resistant to the action of the toxin, they should not knowingly be permitted to ingest such media Since botulinum toxin is quite thermolabile, cooking of garbage at 212° F

for 30 minutes, according to regulations commonly set up for the control of vesicu lar exanthema, should destroy the toxin

The spores on the other hand, have great thermal resistance It apparently varies with the strain and with pH and other conditions of the medium Esty (1923) found them to be among the more resistant of bacterial spores and reported the maximum heat resistance in a phos phate solution of pH 7 0 to be 330 minutes at 100°C, 110 minutes at 105°C 33 minutes at 110°C 11 minutes at 115°C, and 4 minutes at 120°C These intervals represented the time in minutes at which no spores survived Tanner and Dack (1922) found considerably greater sur vival times under conditions of dry heat Freezing has little deleterious effect on either spores or toxin (James 1933)

In view of the ubiquity of *Cl botulinum* and its ability to survive under most natural conditions there seems to be little hope of completely eradicating botulism On the other hand, the disease should be permitted to occur only under uncontrol lable circumstances

REFERENCES

Anderson A A Michener H D and Olcott H S 1953 Effect of some antibiotics on *Clostridium botulinum* Rept West Reg Res Lab Albany N Y 3 521

Bennetts H W and Hall, H T B 1938 Botulism of sheep and cattle in Western Australia Its cause and its prevention by immunization Australian Vet Jour 11 10.

Boroff D A Raynaud M and Prevot A R 1952 Studies of toxin of *Clostridium botulinum* type D Jour Immun 68 503.

Bronfenbrenner J F., and Schlesinger J 1924 The effect of digestive juices on the potency of Botulinus toxin Jour Exper Med 59 509

Buckley, J S and Shippen L P 1917 Preliminary report on the relation of anaerobic organisms to forage poisoning Jour Amer Vet Med Assn 50 809

Burke G S 1923 Dormancy in spores of *Clostridium botulinum* Abst Bact 7 13

Burov A E Denison N J Katz, F M and Segal, M S 1937 Role of animals in spread of botulism Ann Mechnikov Inst 6 27

Coleman G E and Meyer K F 1922 Some observations on the pathogenicity of *B botulinus* N Jour Infect Dis 31 622.

Cook, E W 1945 The occurrence of *Clostridium botulinum* and *Clostridium tetani* in certain plots of soil Ohio State Univ Abst Doctoral Diss 47 9

Dack G M and Gibbard, J 1926a Studies on botulinum toxin in the alimentary tract of hogs rabbits guinea pigs and mice Jour Infect Dis 39 173
——— and ——— 1926b Permeability of the small intestine of rabbits and hogs to botulinum toxin Jour Infect Dis 39 181

Dickson E C 1916 Botulism Proc Soc Exper Biol and Med 14 17
——— 1917 Botulism A cause of limber neck in chickens Jour Amer Vet Med Assn 51 612.
———, and Shevky R 1920-21 Botulism A study of the action of the toxin of *B botulinus* upon the living tissues Jour Soc Exper Biol and Med 18 313

Dolman E C 1957 The epidemiology of meat borne diseases World Health Organization Monograph (Geneva) Meat Hygiene p 82

Esty, J R 1923 The heat resistance of *B botulinus* spores Amer Jour Public Health 13 108

cell walls and some areas of complete necrosis The extent of the applicability of these observations to swine remains to be shown

DIAGNOSIS

Diagnosis of botulism is generally based upon history symptoms, absence of gross lesions and the finding of a suspect source of toxin Occasionally the source of toxin can be demonstrated in the stomach of the animal dead from the intoxication For example, the eating of carrion containing maggots can sometimes be determined at necropsy When this is true, it may lead to the source

Orr, as early as 1921 used mice to test for the presence of the toxin and control mice injected with the then known types of antitoxin to determine the type of toxin present This procedure is still of paramount value in specific diagnosis Kelser (1923) then reported that the complement fixation test could be used to identify the organism and its toxin in culture and in canned foodstuffs

Cl botulinum occasionally may be isolated from animals with botulism, but not with sufficient regularity to be of much value as a diagnostic procedure Therefore negative results do not preclude the possibility of botulism

Burke (1923) found that the spores sometimes go through periods of dormancy outside the body during which they do not vegetate readily — periods as long as 92 days in agar and 144 days in broth This phenomenon may account for negative results when some supposedly spore containing materials are cultured Thus the practical and specific diagnosis of botulism requires (1) consideration of history, clinical signs, and negative necropsy findings, and (2) finding the source of toxin, demonstrating its presence by pathogenicity tests, and typing it by cross-protection tests

TREATMENT

Treatment of botulism depends upon the early use of antitoxin in massive quantities (Guyton and MacDonald, 1947)

However, certain factors may justify modification of this generalization In cases in which large numbers of the bacteria have been ingested along with the toxin anti bacterial therapy may be of some value (Kudo, 1931) Since antibacterial serum is not freely available, one is limited largely to the use of antibiotics effective against Clostridium Among these (Andersen *et al*, 1953) Aureomycin and Terramycin are the most readily available

It has been demonstrated experimentally that botulism intoxication proceeds much more slowly when the subject is under the influence of anesthetics or sedatives, and their use has been suggested to permit more time for the action of the antitoxin to take place Since there is not enough time for typing the toxin, practical therapy involves use of a polyvalent antitoxin Soaps or oils administered by stomach tube or by enema may tend to reduce absorption of toxin and hasten emptying of the tract, however, this or any of the treatments must be administered early for beneficial results

Thus an all out attempt to save a valuable animal might include immediate administration of (1) soaps or oils by stomach tube, (2) broad spectrum antibiotic, and (3) anesthetic or sedative, and maintenance of effective levels of all until (4) the polyvalent antitoxin can be obtained and administered in massive dose and until the outcome is assured

IMMUNITY

While the toxin of *Clostridium botulinum* is immunogenic and may be used to produce not only antitoxins but also toxoids (Sterne and Wentzel, 1950), the use of such products in swine is not practical, even in garbage feeding enterprises It is rendered impractical by cost and the low incidence of the disease which, in turn, is apparently due to the natural resistance of swine to botulism

EPIZOOTIOLOGY AND CONTROL

Since botulism is an intoxication, occurring usually as a result of ingestion of preformed toxin, transmission does not

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Edema Disease

Edema disease of swine is also known as enterotoxemia, gut edema, gastric edema, and edema of the bowel No other animal species is known to exhibit the same set of circumstances, symptoms, and pathological changes which characterize the disease in swine

The condition was first described by Shanks (1938) as occurring in the swine raising areas of northern Ireland, where it had been observed since 1932 Since that time it has been reported from countries such as England, South Africa, Norway, Holland, Canada, the United States, France, Denmark, and Sweden and can probably be recognized as having world wide distribution, occurring in any area of swine production Considerable investigational work has been carried out in Ireland and re ported by Lamont et al (1950), Timoney (1950), and Lamont (1953)

Edema disease is important because it is quite widespread and has a high incidence of occurrence, especially in areas where the swine population is high It is not a seasonal disease since it has been observed during every month of the year Because of the high rate of incidence, the economic loss to the swine industry is considerable Another factor in the importance of edema disease is the striking similarity of some of its pathological changes to those of other serious swine diseases Bennett (1954) reported on cases of edema disease

which would have been diagnosed as chol era on the basis of history, symptoms, and gross lesions except that the presence of cholera virus could not be demonstrated by pig inoculation tests

ETIOLOGY

The exact cause of the condition has not been fully demonstrated, although the work carried out in Ireland by Timoney (1950) strongly indicated that the condition was a toxemia Vesselinovitch (1955) investigated the serum proteins of pigs affected with edema disease He could divide the cases studied into two groups on the basis of the presence or absence of a clinically observable edema Both groups showed a decrease in the percentage of serum albumin The group showing clinical edema gave higher than normal values for both alpha and gamma globulins, and the group without clinical edema gave higher values for gamma globulins only He stated

The lack of an alteration to the protein pattern of sufficient specificity to imply that the changes stemmed from a particular organ or organ system makes it impossible to state the exact site and nature of the disturbances re sulting in this dysproteinemia However it seems possible that the dysproteinemia in these cases is an instance of tissue proteinuria being the manifestation of a non specific re action of the body to stress accompanied by an enhanced activity of the reticulo-endothelial system

[495]

Graham R Bruckner A L and Pontius R L   1917   Studies in forage poisoning VI by Agr Exp Sta Bull No 207

Guyton A C and MacDonald M A   1947   Physiology of botulinus toxin Arch Neurol and Psychiat 57 578

Hagan W A and Bruner D W   1957   The Infectious Diseases of Domestic Animals 5th ed Comstock Publ Assoc Ithaca N Y

Hall, L C and Stiles G W   1938   An outbreak of botulism in captive mink on a fur farm in Colorado Jour Bact. 36 982

Hamre D H   1941   The properties and the action of botulinus toxin Univ of Colo Stud Ser A 26 86

Hart G H   1970   Botulism in chickens Jour Amer Vet Med Assn 57 75

James L H   1933   Effects of freezing on the spores and toxin of *Cl botulinum* Jour Infect D s 52 236

Keiser R A   1923   The identification of *Bacillus botulinus* and its toxin in culture and in canned foodstuffs by serological methods Amer Jour Public Health 13 366

——— and Schoening H W   1948   Manual of Veterinary Bacteriology 5th ed Williams and Wilkins Co Baltimore Md

Kudo Y   1931   Experimental studies on the pathogenesis of botulism Jap Jour Exper Med 9 373

Merchant I A and Packer R A   1956   Veterinary Bacteriology and Virology 5th ed Iowa State College Press Ames Iowa

Nelson C I   1927   The relationship between the intracellular globulin and the toxin of *Cl botulinum* Jour Infect D s 41 9

Orr P F   1921   A rapid method of determining the presence and type of botulinus toxin in contaminated foods Jour Infect Dis 29 287

Quin J I   1946   The biological action of botulinus C (Lamsiekte) toxin So African Jour Sci 42 157

Quortrup E R and Holt A L   1941   Detection of potential botulinus toxin producing areas in western duck marshes with suggestions for control Jour Bact 41 363

Scheibner von G   1935   Die Empfanglichkeit des Schweines fur Botulinustoxin der Typen A E Deutsch tierarztl Wschr 62 355

Schmidt H   1930   Lo n disease of cattle Tex Agr Exp Sta Rept p 7

Shay D E   1916   An histologic study of effects of experimental botulinus poisoning on the liver of guinea pigs Jour Clin Invest 25 687

Sherman J M Stark C N and Stark P   1927   The destruction of botulinum toxin by intestinal bacteria Proc. Soc. Exper Biol and Med 24 546

———, and ———   1928   Destruction of botulinum toxin by milk bacteria Jour Dairy Sci 11 352

Starin W A   1925   Pathogenicity of *Clostridium botulinum* Jour Infect Dis 36 383

Sterne, M and Wentzel L M   1950   A new method for the large scale production of high titer botulinum formol toxoid types C and D Jour Immun 65 175

Sutherland D N   1955   Botulism in farm animals Queensland Agr Jour 81 97

Swab C M and Herbert F G   1933   The ophthalmic lesions of botulism Additional notes and research Brit Jour Ophthal 17 129

Sysak N and Meniowitsch K   1929   Zur patholog schen Veranderung beim experimentellen Botulismus Frankfurter Zeitschr f Path 38 561

Tanner F W and Dack G M   1922   *Clostridium botulinum* Jour Infect Dis 31 92

Theiler A   1927   Lamsiekte (Parabotulism) in cattle in South Africa 11th and 12th Rept Part II Dir Vet Ed and Res Dept of Agr Union of South Africa

Van Ermengen E.   1897   Ueber einen neuen anaeroben Bacillus und seine Beziehungen zum Botulismus Zeitschr f Hyg u Infektionskrankh 26 1

they may be observed in different individuals

In areas where hog cholera is not prevalent the weak incoordinated gait is said to be characteristic The progressive development of clinical signs of edema disease occurs at a fairly rapid rate and in the usual course of events either mortality or a change toward recovery occurs within 48 hours Only occasionally do affected individuals survive for as long as 5 to 7 days

PATHOLOGICAL CHANGES

After a number of necropsies the observer may gain the impression that the lesions to be found in cases of edema disease are quite variable Actually since the lesions are either edema or hemorrhage the variation occurs in the matter of location and severity of either or both of these lesions It is quite common to find both edematous and hemorrhagic lesions in the same individual Occasionally cases appear in which there is only one or the other of these two types of lesions

Most of the names by which the disease is known refer to and are descriptive of only the edematous lesions During the early years of the recognition and study of the disease the most prominent pathological

change was a layer of edema in the stomach wall Other locations in which edema may be rather prominent include the mesenteric folds of the coiled portion of the large intestine (Fig 34 2) the eyelids the ears subcutaneous tissues of the face and jowls and the ventral and ventro lateral areas of the abdominal wall

The gastric edema is most often found along the greater curvature usually in the cardiac portion but it may extend into the fundic and esophageal areas It consists of a layer of jelly like edema between the muscular and mucosal layers It may be either clear and practically colorless or blood tinged and red colored It may vary in thickness from a barely visible layer to one of about an inch in thickness The lesion also varies in size from an area of about 4 inches in diameter to a small localized spot of less than 1 inch in diameter An incision along the greater curvature is not always sufficient to demonstrate this edematous stomach lesion The smaller localized areas are sometimes found only by making several cuts directed from the greater curvature toward the esophageal opening

Edema of the mesenteric folds of the coiled large intestine also varies in amount

FIG 34 2 — Edema of the large intestine showing the characteristic increase of fluid in the tissues in the folds of the colonic coil (see arrows) (Photograph by H W Dunne)

In addition to providing evidence that the disease is a toxemia, Timoney's 1950 work also indicated that the probable toxic principle could not be neutralized with pulpy kidney disease antisera This would indicate that the toxin of *Clostridium perfringens*, type D, commonly involved in sheep enterotoxemia, is not a significant factor in edema disease

Field and Gibson (1955) showed that *Cl perfringens*, type C, was the causative agent of a disease causing loss in baby pigs However this disease has certain characteristics which are not common in edema disease, and it is believed that toxemia due to *Cl perfringens*, type C, should not be considered in the edema disease syndrome

Experimental procedures and a study of natural outbreaks of edema disease tend to support the theory that the disease is a toxemia It is characteristically irregular in the manner in which it affects individuals and droves In natural outbreaks, the random scattering of individual animals which become affected on a single farm, as well as a similar random scattering of farms on which the disease occurs in an area, does not follow the pattern shown by known infectious diseases Careful experimental work has failed to give evidence that the disease can be successfully reproduced with any infectious agent

Timoney (1950) reported some success in reproduction of the disease by means of injection into pigs of centrifuged supernatant fluid from either diluted or undiluted intestinal contents of naturally occurring cases Timoney (1956) reported that such supernatant fluid retained its toxicity after being stored at a temperature of approximately 20° F for 11 months He also indicated that heating at 60° C for 15 minutes might not inactivate the toxin

Gregory (1955) reported limited evidence of success in reproducing edema disease by means of a toxin or toxins associated with hemolytic coliform organisms Timoney (1956) presented more conclusive evidence that toxic material associated with a hemolytic coliform organism was of significance in causing edema disease A neutralizing antiserum could be developed in pigs injected with suitably treated material obtained from the supernatant fluid of centrifuged intestinal contents of naturally occurring cases, and a similar protective serum could be obtained from pigs immunized with a hemolytic coliform organism

CLINICAL SIGNS

Edema disease is often a rapidly acting toxemia, and symptoms are not always observed, especially at the beginning of an outbreak The early observable clinical signs consist of a mild listlessness, a little reduction in appetite, and a weak, wobbly incoordinated gait The pigs may show aimless walking or walking in circles, and an apparent blindness exhibited by running into objects A gradual paralysis develops rapidly, and many pigs show fine generalized muscular tremors These central nervous disturbances often develop into convulsions, and the pigs may become prostrate with their legs in either constant or intermittent running motions (Fig 34 1) Very few recoveries have been reported after these severe signs have developed Recovery of less severely affected individuals appears to be of common occurrence

Most affected animals do not show an increase in temperature, but a few individuals may be found with temperatures of 104-105° F Both diarrhea and constipation have been observed, but it is not clear whether one of these signs precedes the other in the same individual, or whether

FIG 34 1 — Central nervous system disturbance in a pig suffering with edema disease (Photograph by H W Dunne)

both, may show extensive hemorrhage In some instances practically all of these various types of hemorrhage can be found in the same individual Hemorrhages of the spleen and kidney are quite common, and in more than a few cases they exactly resemble those of cholera

A rather commonly observed indication of renal hemorrhage is the presence of a very few small and inconspicuous petechiae on the exterior surface, with several prominent petechial and ecchymotic hemorrhages on and in the papillae Some of the kidneys with little external evidence of hemorrhage will contain small amounts of free blood in the calyces and pelvis

Another common observation of the cut surface when the kidneys are cut longitudinally into halves is that the renal cortex tends to be relatively low in blood supply while the medullary portion tends to be engorged with blood Occasionally this abnormal distribution is quite marked, leaving the cortex definitely ischemic and the medulla highly congested and hemorrhagic Even in instances of more mildly altered blood distribution, the longitudinally cut surface presents a zoned appearance due to the congestion of the medullary portion of the kidney

In a few instances the capsule of the kidney may be thick and edematous and separated from the kidney by a considerable amount of blood tinged fluid In these cases the kidney appears quite ischemic and the fluid itself gels upon exposure to the air, similar to the gelling of fluids found in the peritoneal and pleural cavities

The mucosa of the urinary bladder may be only mildly congested The congestion may appear as scattered patches it may cover the entire surface, or it may present a streaky appearance The mucosa may show a few or many petechiae and ecchymoses or the entire surface may be severely hemorrhagic, in which case the serosal surface may also be entirely hemorrhagic and the bladder wall is much thickened

The lymph nodes, in addition to being swollen and edematous often show hemorrhagic lesions Any or all of them may be so affected The hemorrhage may be either peripheral, diffuse, scattered petechial, or ecchymotic, or the entire node may present a solid hemorrhagic appearance even when cut in any plane All of these variations of hemorrhagic lymph nodes may be seen in different nodes of the same individual Irregularly shaped areas of hemorrhage may be observed on the surface of the liver However the liver is the organ in which grossly visible hemorrhages are most rarely found The gall bladder may be edematous or its mucosa may show petechiae and ecchymoses Hemorrhages can also be found in the subcutaneous tissues, the muscle, and the serous membranes of the legs, especially around the leg joints the meninges the lining of any or all of the sinuses of the skull, and the serous covering of the nasal septum

DIAGNOSIS

Some of the common features to be observed during the course of an outbreak of edema disease are important diagnostic aids It is a disease which requires the veterinarian's close attention to an accurate record of events noted by the owner, plus close observation of occurrences after the owner brings it to his attention In some cases a good history may be the decisive factor in arriving at a diagnosis The first of these rather common features is the tendency for the disease to appear quite suddenly without previous warning One or more animals may be found dead, although the owner may be sure that the animals appeared to be normal only a few hours earlier Second, the disease usually affects the thriftiest, fastest growing individuals in the drove Third, there is a high mortality in the visibly affected animals and the duration of the disease is usually short, although some pigs may survive for as long as 5 to 7 days As a final significant feature the losses may stop as suddenly and unexpectedly as they appeared A few pigs may show sufficient edematous swelling of the eyelids, ears, and face to be of clinical diagnostic importance

from a barely visible layer to a very conspicuous accumulation In some cases a layer of jelly like edema can be found surrounding the rectum The edema of the eyelids ears and subcutaneous tissues of the face often produces a swelling that is observable on clinical examination Practically all of the lymph nodes are edematous and show some degree of enlargement

Lung edema is not uncommon and in a few instances the edema together with the concurrent congestion and hemorrhage, closely resembles that seen in cases of sheep enterotoxemia and may be the only prominent lesion found at necropsy Edema of the brain occurs and is probably responsible for some of the signs of central nervous system origin that occur during the course of the disease

Another common necropsy finding is the presence of varying amounts of fluid in the pericardial sac, the pleural cavity, and the peritoneal cavity This fluid may be either clear, colorless golden straw colored, or more or less blood tinged It usually coagulates or gels rapidly upon exposure to the air

Schofield (1953) suggested that the name be changed to enterotoxemia, stating that edema was only one manifestation of the toxemia and that it was not always present in every individual affected in a typical outbreak Even before that time however, the early investigators had pointed out that the disease was probably a toxemia and that numerous hemorrhagic lesions could be found

In some individuals such hemorrhagic lesions are very prominent and widespread in cases of edema disease The early reports include special names for the hemorrhagic lesions found in two different locations a flea bite skin lesion and a mulberry heart The flea bite lesion refers to a cutaneous hemorrhage which is usually small in size but numerous and resembles actual flea bites This lesion is most easily seen on the thin haired ventral body surface and the medial surfaces of the rear legs but it may vary in size and cover a much more extensive portion of the body

It is not a common lesion, and may be seen only occasionally

The mulberry heart is a severe and diffuse hemorrhage of the epicardial and myocardial tissues Again, this severe cardiac hemorrhage is not seen frequently, however, less severe cardiac hemorrhages are rather common In some cases these less severe hemorrhages appear as superficial surface streaks and in other cases they are more diffuse and not so sharply delineated as streaks Subendocardial hemorrhages of the left ventricle are quite common

Gross hemorrhages can be found in numerous locations in the body They may be seen as either petechiae or ecchymoses in the larynx trachea, and lungs Sometimes large, irregularly shaped, but sharply circumscribed areas of lung tissue may be seen to be hemorrhagic on cut surfaces with only a very small or no external surface area to indicate their presence Other affected lungs contain many small, slightly diffuse areas of hemorrhage scattered throughout the organ and visible on both external surfaces and cut surfaces The mucosa of the stomach may be congested and hemorrhagic in any degree from slight to severe, and these hemorrhagic lesions may be found overlying relatively thick layers of edema Petechiae, ecchymoses and diffuse areas and streaks of hemorrhage may be found on the serosal surface of the stomach The intestinal mucosa is a very common site of hemorrhagic lesions Either the small or large intestine, or both, may show scattered petechial or ecchymotic hemorrhages Hemorrhagic streaks along the tops of longitudinal folds of mucosa may be observed Large but variable-sized patches of hemorrhage may be scattered along a considerable length of intestine These patches can be seen through the serosal surface as dark red, irregularly shaped areas A considerable length of intestinal mucosa may be uniformly congested and hemorrhagic, and the lumen contents may be quite bloody in appearance

In severe cases the serosal surface of either the small or large intestine, or of

purpose is not yet available and must await the development of further information concerning the cause or causes of edema disease

Differential blood cell counts and histopathologic examination of brain tissue can be used to differentiate many cases of cholera and edema disease Edema disease does not produce the marked leukopenia or the encephalitic lesions found in many cases of cholera These procedures, how ever, especially the histopathologic examination, are special techniques which are not readily available in at least two situations where diagnostic decisions must usually be made without benefit of laboratory procedures

The first of these situations would be routine field necropsies for diagnostic purposes where it is quite easy to make a wrong diagnosis of hog cholera on the basis of symptoms and hemorrhagic lesions The second situation involves the examination of carcasses and viscera for food purposes Several instances have been observed in which meat inspection procedures have shown significant petechial and ecchymotic hemorrhages of the kidneys and urinary bladder together with peripheral congestion and hemorrhage of various lymph nodes In one instance, inspection prior to slaughter had shown no abnormalities either in temperature or in actions The pigs had grown very rapidly, reaching a weight of 200 lbs when they were approximately 18 weeks of age While only the hemorrhagic lesions were noted, careful examination of the stomach wall might have shown the presence of edema in a very small percentage of these cases Continued observation of the drove from which these suspicious cases originated failed to show any significant clinical evidence of the presence of edema disease or any infectious disease Further study of the significance of such findings in food inspection situations will need to be made before probable errors of both economic and biological consequences can be avoided

Erysipelas is another common swine dis ease which can resemble both edema disease and cholera However, differentiation can be made on the basis of bacteriologic examination with recovery of the causative organism Some care should be used in selecting a suitable specimen for bacteriologic examination in order to avoid the use of material from animals which have received large or repeated treatments of antibiotics or antisera within several hours of the time the examination is made If specimens from swine which have received treatment are used, inconclusive negative results may be obtained

Edema disease must also be differentiated from African swine fever This serious swine disease is not known to occur in any area outside of the African continent, and every precaution is taken to prevent its introduction into other localities The outstanding pathological changes observed in African swine fever are reported to be hemorrhages due to damage done by the causative virus to the endothelial cells of small blood vessels Marked edema of the lungs, spiral colon, cecum, and mesentery is also reported in some cases of African swine fever

The complete report of lesions observed in African swine fever presents a picture which is practically identical to the changes observed in some of the more severely hemorrhagic cases of edema disease On the basis of a report on African swine fever made by the Committee on Exotic Diseases, United States Livestock Sanitary Association (1954) some differential features appear to be applicable

Although there is a high mortality rate with African swine fever, death usually does not occur so rapidly as in the case of edema disease Death from the virus disease usually occurs on the fourth to seventh day following development of symptoms while mortality from edema disease usually occurs around 48 hours or less A temperature increase to 104° F and up to 108° F is expected in African swine fever, but no rise in temperature is ordinarily expected in edema disease At least one lesion ap-

The weak wobbly incoordinated gait is an important symptom however, it is a common sign of both edema disease and hog cholera Appetite is one indication which is often used clinically for diagnosis of swine diseases In edema disease the appetite may or may not be affected However, if the case terminates fatally, its duration is usually sufficiently short that upon necropsy the stomach will be found to contain considerable food material The same thing can be said of certain cases of cholera Many pigs affected with cholera show only a very brief period of decreased appetite, and unless they are being closely watched this symptom may not be observed

Age, another factor used to differentiate some swine diseases is unfortunately not helpful in dealing with edema disease Early studies of the disease indicated that it commonly occurred in pigs which were from 8 to 14 weeks of age As more evidence and experience accumulated, it was recognized that the disease might affect swine of any age from only a few days to a year or more Typical edema of the colonic mesentery has been observed in pigs as early as 21 to 36 hours after birth

Body temperature is another useful clinical sign for diagnostic purposes A majority of the animals affected with edema disease do not show a higher than normal temperature, but a few individuals are found with fevers as high as 105° F Prior to 1950 this clinical sign might have been considered a reasonably good basis for differentiation between edema disease and hog cholera With each passing year, however, more and more cases of hog cholera have been observed in which body temperature has paralleled that of edema disease In many instances edema disease outbreaks encourage an incorrect diagnosis by appearing very shortly after the pigs have been vaccinated against hog cholera

Although edema and hemorrhage are nonspecific lesions frequently seen in other diseases of swine, most cases of edema disease present a combination of both edematous and hemorrhagic lesions Under these conditions satisfactory evidence can be accumulated by careful and complete observation and evaluation so that a reasonably accurate decision can be reached Where so much variation in findings exists, experience is the best teacher, and it will be quite advantageous to take every opportunity to perform complete and careful necropsies All features of the history of the case should be taken into consideration when the necropsy findings are evaluated

The edema sometimes found in the jowl and throat area must be differentiated from that found in anthrax, malignant edema and chronic multiple infection of the tonsilar areas Such infections can be readily demonstrated by bacteriological methods Cases may be encountered in which sufficiently satisfactory edematous lesions cannot be found Hemorrhagic lesions alone are much more difficult to evaluate for differential diagnostic purposes because they are quite common in both toxic and infectious conditions The extreme and massive hemorrhages that are sometimes observed are considered more characteristic of toxemias than of septicemias The less severe hemorrhages, however, pose quite a problem and a few infectious conditions as well as toxemia must be considered as possibilities In areas where hog cholera has not been eliminated, it is the infectious disease to be most frequently considered

In the present state of our knowledge of both cholera and edema disease, it is not always possible to differentiate them on the basis of gross lesions alone In a few instances even a good history together with observation of gross lesions will fail to provide enough evidence for differentiation A positive diagnosis of cholera can be made by animal inoculation tests, however, such a procedure is so expensive and time consuming that it has no practical value for the case under consideration Even if the presence of cholera virus can be demonstrated in this way, the possibility of simultaneous occurrence of both cholera and edema disease would need to be considered A completely satisfactory procedure for this

REFERENCES

Bennett, P C 1954 Edema disease of swine Iowa Vet 25 (No 6) 9

Field, H I, and Gibson, E. A 1955 Studies on piglet mortality 2 *Clostridium welchii* infection Vet. Rec. 67 31

Gregory, D W 1955 Role of beta hemolytic coliform organisms in edema disease of swine Vet Med 50 609

Lamont, H G 1953 Gut edema in pigs Proc 90th Ann Meet Amer Vet Med Assn p 186

———, Luke, D, and Gordon, W A M 1950 Some pig diseases Vet Rec 62 737

Schofield, F W 1953 Should the name oedema disease be changed to enterotoxemia of swine? Vet. Rec. 65 143

——— and Schroeder J D 1954 Some important aspects of oedema disease in swine (enterotoxemia) Canad Jour Comp Med 18 24

Shanks, P L 1938 An unusual condition affecting the digestive organs of the pig Vet Rec 50 356

Shipp W L, Burnside, J E, and Atwood M B 1953 A disease of swine and cattle caused by eating moldy corn Proc 90th Ann Meet Amer Vet Med Assn p 174

Timoney, J F 1950 Oedema disease of swine Vet Rec 62 748

——— 1956 Oedema disease of swine Vet Rec 68 819

United States Livestock Sanitary Association 1954 Report of the Committee of Exotic Diseases p 15

Vesselinovitch, S D 1955 Electrophoretic studies of oedema disease in swine Brit Vet Jour 111 398

pears to be of significance In African swine fever the spleen is reported to be frequently dark red and enlarged to as much as twice the normal size In edema disease the spleen is never enlarged, and in at least 50 per cent of the cases there is a noticeable lightening of color from the normal red appearance

A conclusive differential diagnosis depends upon the reproducing of African swine fever by inoculation of susceptible swine, including hog cholera immunized and hyperimmunized individuals, with fresh material such as blood or spleen from the suspected case

Another toxemia of swine that possibly could be confused with some of the very severe hemorrhagic cases of edema disease is poisoning by Warfarin Pathological changes in cases of Warfarin poisoning are primarily hemorrhage and edema The hemorrhages are of the massive whole blood type similar to those seen in cases of sweet clover poisoning in cattle They are most prominent in the lower portions of the body such as the ventral abdominal wall and the legs Some cases of edema disease have very noticeable hemorrhages in these same parts of the body, but they are not the typical extravasation of whole blood seen in Warfarin poisoning

A disease of swine has been caused by ingestion of moldy corn has been reported by Sippel *et al* (1953) The lesions described as occurring in this disease appear to be identical in many respects to those seen in severely hemorrhagic cases of edema disease Differentiation can be made on the basis of consumption of badly molded corn by the affected pigs Such badly molded feed is seldom, if ever, associated with edema disease Another point of some differential value is the commonly observed presence of subperitoneal and thoracic cavity hematocysts in moldy corn disease as compared with the absence of such whole blood accumulations in edema disease

TREATMENT

Due to the fact that knowledge concerning the cause of the disease is incomplete, the most effective treatment and preventive measures have not yet been developed Practically everything from magnesium sulfate to hog cholera serum has been used in treating cases of edema disease Any treatment will at times apparently give good results but such results can never be demonstrated consistently

The so called broad spectrum antibiotics have been used in numerous cases of edema disease They undoubtedly produced some effect on the bacterial flora of the digestive tract when given orally, and in some instances the effect produced was very probably beneficial However, it must be remembered that the causative agent or agents of edema disease are not fully understood, and there may be equal probabilities that the effect produced by the antibiotics could be either injurious or negative Products such as mixed bacterins, erysipelas antiserum and cortisone have at times received credit for beneficial results in edema disease, but conclusive proof of such value is lacking Over a period of several years the most satisfactory line of treatment and management has been the use of a saline cathartic and a withholding, or very sharp reduction in amount of feed for a short but variable length of time Such treatment is nonspecific, but there is logical argument in favor of its use in cases of enterotoxemia

Timoney s (1956) announcement of considerable success in pinpointing the probable cause, together with very promising immunologic studies, creates considerable hope that much more specific treatment and immunologic preventive measures will be developed in the near future

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Fungous Toxins

Moldy Corn Poisoning

Poisoning of livestock by fungous toxins
has long been suspected Moldy corn
poisoning of horses and moldy hay and
grain poisoning of various animal species
are illustrations There are several other
types of affections caused by molds, but
these are not of a strictly toxin' nature
In Russia, Gadjusek (1953) has reported
the poisoning of people and horses by
mold toxins on wheat allowed to over
winter beneath the snow Frohner and
Voelker (1950) and Glasser et al (1950)
report mold intoxications in swine and
other animals Christensen and Kern
kamp (1936) reported intestinal dis
turbances in swine eating moldy barley
Forgacs et al (1954) and Forgacs and
Carll (1955) have shown molds to be
toxic for calves and poultry Sippel et al
(1953) described moldy corn poisoning
in swine and cattle The chemical nature
of these toxins has not been determined

Poisoning from moldy corn peanuts,
bread, and oats has been observed by the
author in Georgia, Florida, and Alabama
Moldy corn poisoning was prevalent in
North Carolina following a hurricane in
the fall of 1955 Case (1956) reported
the same type of mold poisoning in Mis
souri and southern Iowa The author has
also heard of mold poisoning in Iowa

from corn kept for several weeks in wagons
before being fed to swine

Forgacs (1952) isolated pure cultures
of Penicillium rubrum (Stoll) and As
pergillus flavus (Link) from corn in fields
in Georgia with which Burnside (1953)
was able to produce acute poisoning in
experimental pigs

ETIOLOGY

The following molds have been incrim
inated as producing acute intoxication
in animals Penicillium rubrum (Stoll)
and Aspergillus flavus (Link) in swine
(Burnside, 1953) and Aspergillus cheva
lieri, A clavatus, and A fumigatus in
calves (Carll et al , 1955) Other fungi have
been involved in the poisoning of other
animals

These fungi are readily cultivatable on
ordinary media Sabouraud s agar, myco
logical agar (Difco), and mycophil agar
(BBL) also serve well

CLINICAL SIGNS

Pigs affected with moldy corn poisoning
exhibit acute and chronic syndromes In
droves in which the acute disease appears,
the animals are found dead or are sick
only two days or less They are depressed
are off feed, and exhibit signs of weakness
by staggering in the hindquarters The
mucous membranes are pale and tempera

Histopathological changes are most prominent in the liver, where in acute cases lesions of acute toxic hepatitis are found Depending on the amount of toxin ingested and length of time involved, the changes vary from fatty degeneration to necrosis with hemorrhage in the lobule In chronic cases, the changes are those of subacute toxic hepatitis There is more or less cirrhotic proliferation of the capsule of Glisson, with accompanying bile duct proliferation Hyalinoid granules of degenerated cytoplasm are frequently seen within hepatic cells Various stages of connective tissue replacement of the centrally located necrotic hepatic cells are seen In many chronic cases examined at necropsy

FIG 35 2 — Hemorrhages in thoracic cavity and axillary space of experimental pig with moldy corn poisoning

there is marked regeneration and hypertrophy of the peripheral hepatic cells in the lobules

Kidney lesions include glomerular atrophy, tubular dilatation, and various stages of degeneration of the epithelium of some medullary rays

Changes in other organs are not remarkable

DIAGNOSIS

This condition may be suspected if pigs are eating molded grain especially corn or peanuts or feeds containing these substances In many cases corn will be found to be on the ground and pigs forced to eat grain they would have avoided had better feed been available

It is well known that all molds are not toxic and that large quantities of moldy feed are eaten without apparent harm Therefore, the mere finding of molded feed is not sufficient for a diagnosis

Necropsy lesions when coupled with the finding of moldy feed have enabled veterinarians in endemic areas to make a diagnosis of this condition with confidence In acute cases, the subendocardial hemorrhages and other larger and smaller hemorrhages have been helpful In chronic cases, which are the most numerous the subendocardial hemorrhages icterus and cirrhosis are seen

A definite diagnosis cannot be made without both the identification of a toxic mold in the feed in sufficiently large quantity and the reproduction of the disease in swine by means of pure cultures of the mold This is a complicated time consuming laboratory procedure that is not practical in routine laboratory diagnostic work

Differential diagnosis should include consideration of those diseases causing icterus, cirrhosis, anemia, and numerous hemorrhages Such diseases are leptospirosis, eperythrozoonosis, coal tar pitch poisoning, and poisoning by some plants, depending on the region Poisoning by Crotalaria spp in hogs is characterized by

tures are normal. Blood may be passed from the rectum. Occasionally central nervous system symptoms are seen, such as standing with the head in a corner or pressing the head against a wall.

Chronically affected animals are depressed, sometimes walk with a stiff gait, have poor appetites, and often stand apart from the rest of the drove with their heads down, backs arched, and flanks tucked up. Their temperatures are normal. Farmers sometimes complained that their pigs were losing weight in spite of ample corn in the field. The mucous membranes of chronic cases were usually icteric.

NECROPSY LESIONS

Large hemorrhages resulting in anemia are a prominent feature of most acute cases, especially in those animals found dead. Large retroperitoneal hemorrhages extending from the diaphragm to the pelvic inlet may be seen. These include large amounts of blood around the kidney and some in the hilus of the stomach (Fig 35 1). In other animals, large amounts of free blood are found in the abdominal or thoracic cavities (Fig. 35.2). Ecchymotic to petechial hemorrhages are sometimes found scattered on serous surfaces. Subcutaneous hemorrhagic areas on the anterior surface of the thigh, in the subscapular region, and on other muscles are not uncommon. Free blood is often found in the intestine. The liver sometimes has petechial or ecchymotic hemorrhages beneath the serosa. The spleen is usually normal, but sometimes has dilated surface capillaries or hemorrhagic infarcts. Epicardial and profuse endocardial hemorrhages are a constant lesion in acute cases.

In chronically affected animals, icterus of the carcass and cirrhosis of the liver are prominent lesions at necropsy. The degree of these changes varies widely. The abdominal or thoracic cavities often contain large amounts of clear, straw-colored fluid. Mitchell *et al.* (1956) have described cases they attributed to mold poisoning that presented "voluminous amounts of a serous exudate within the thoracic cavity, accompanied with extensive pulmonary edema and distension of the interlobular septa with edema." Cirrhosis was not present in these cases. Gelatinous infiltration beneath the serosa of the colon is sometimes observed in chronic cases. The kidneys are often pale and swollen Lymph nodes are congested and "watery" in many cases. The ecchymotic hemorrhages on the muscles, anterior surface of the thigh, and subscapular region, as seen in acute cases, are also seen frequently in chronic cases These lesions are similar to those found in hemorrhagic disease of poultry. Hemorrhages beneath the endocardium are almost a constant lesion in chronic cases.

FIG. 35.1 — Lesions in pig naturally affected with moldy corn poisoning Large retroperitoneal hemorrhage surrounding kidney and in hilus of stomach

The prolapse may evert a distance of 6 inches with a diameter of 4 inches Rubbing of the exposed portions may cause injury and infection Prolapse of the rectum may occur secondarily in 5 to 10 per cent of the cases Vaginal prolapse was seen in up to 30 per cent of the Iowa cases An unusual amount of prepucial inflammation was noted in males and some mammary enlargement in gilts Gilts from 6 weeks old up to 100 or 150 lb in weight are most often affected Older animals appear resistant Death may result from hemorrhage infection uremia or infection of the urinary tract McNutt et al (1928) noted no effect on subsequent breeding efficiency of affected gilts

PATHOLOGICAL CHANGES

Lesions are confined to the external genitalia and consist of edema congestion and hemorrhage Secondary septic inflammatory changes are often present and may ascend the internal urinary tract

DIAGNOSIS AND TREATMENT

Normal estrum or injury to the external genitalia are the only things likely to be confused with this condition Experimental cases require 4 to 6 days to develop symptoms when fed moldy corn or barley Recovery from the swelling follows in 7 to 10 days after removal of the damaged grain There is no specific treatment other than a change to sound feed

EPIZOOTIOLOGY

An outbreak occurred in Iowa in 1926 (McNutt et al, 1928) following the wet test September on record to that time Both white and yellow dent corns were involved The condition was directly proportional to the percentage of damaged corn in various sections of Iowa that year and was also seen in several other mid western states

This is another condition emphasizing the potential danger of molded or damaged grain

Ergotism

Ergotism is probably a rare disease in swine in the severe gangrenous form but may be prominent in the cause of agalactia birth of small weak short lived or dead pigs

ETIOLOGY

This condition is caused by the sclerotium of *Claviceps purpurea* which contains the toxic levulorotatory alkaloids ergotoxine and ergotamine This fungus is usually found growing on the seed heads of Dallis grass (*Paspalum dilatatum*) rye and other small grains

CLINICAL SIGNS

Frohner and Voelker (1950) indicate that the pig is relatively resistant to this type of poisoning and cite the case of a pig that was fed 11 kg of the fungus over a 2 month period before succumbing

The most serious loss in swine probably occurs in sows that are chronically affected and develop the agalactia syndrome re-

ferred to above Nordskog and Clark (1945) in their experiments fed rations containing 0 5 1 0 and 3 0 per cent ergot Animals on all rations showed almost complete lack of udder development and failed to secrete milk at farrowing Nine sows averaged about 9 pigs of 1 8 lb each at birth of which about half were born alive but died shortly after birth Control sows had the same sized litters that averaged 2 9 lb each, of which only 3 were born dead Milk secretion was normal Burns (1953) has indicated that the classical gangrenous type of ergotism with dry gangrene of the ears extremities and pieces of skin of the trunk occurs in the pig

In addition Frohner and Voelker (1950) list gangrene of the claws ends of the phalanges metacarpi and metatarsi the tail and nipples Limping and inability to rise are also seen

Although some of the sows studied by

sudden death in the acute form and loss of appetite unthriftiness, and anemia in the chronic form Sudden deaths are directly attributable to gastric hemorrhage (Emmel and Sanders, 1942) Dicoumerin rat poisons such as Warfarin [*] might cause lesions that could be confused with acute cases of moldy corn poisoning

The necropsy lesions listed above should be sufficient to differentiate these conditions

TREATMENT

No specific treatment is known The pigs should be taken off the moldy feed immediately In order to detect chronically affected pigs that will not make profitable gains, the pigs can be placed on a good ration for two weeks At the end of that time those not responding are sold for slaughter

EPIZOOTIOLOGY AND CONTROL

This condition has appeared in the South predominantly on early soft corns

planted for "hogging off' However, it has also been seen on hard, later maturing varieties In other sections it has been seen when corn is stored under conditions allowing it to become moldy

In order to keep swine from eating moldy corn, temporary fences have been used so that small sections of a field can be eaten out completely before any ears that have been knocked to the ground by the pigs have had an opportunity to become molded The fences are moved when indicated

As a means of determining if molded feed is safe for consumption, an animal of low value can be fed the grain in question for two weeks and observed for toxic symptoms If none are observed, the calculated risk of feeding the moldy grain can then be taken The method is not entirely safe, due to the danger of a chronic intoxication producing liver damage that could result in slow gains or delayed symptoms of a more serious nature

Vulvovaginitis

This condition has been reported by McNutt et al (1928), Pullar and Lerew (1937), and by McErlean (1952) respectively from Iowa, Australia, and Ireland So far as is known, only swine are susceptible

ETIOLOGY

The disease has been produced experimentally with 'spoiled and molded corn" (Iowa), moldy barley (Ireland), and moldy maize (corn) (Australia) McErlean identified Fusarium graminearum (conidial stage of Gibberella zeae) ' and a species of Cephalothecium ' in the moldy barley with which he worked The Australian workers considered the active principle to be eliminated in the urine, producing an irritant effect on the vulva McErlean postulated that " the substance is more likely to be of the nature

of an estrogen which is absorbed from the alimentary canal and operates systemically '

CLINICAL SIGNS

McNutt et al (1928) very ably describe the condition as follows

The first change to be noted is a gradual enlargement or swelling of the vulva It seemingly differs in no way from enlargement of the vulva due to the heat period, but the swelling continues until the vulva is smooth very firm tense and elevated or swollen out from the body Then it is that the lips separate and the vaginal mucosa, only slightly injected or reddened begins to show The inner portions of the vulva and vaginal mucosa continue to swell until the mucosa protrudes through the lips of the vulva The weight of the prolapsed portion drags the more anterior portions out Return circulation is partly checked resulting in passive congestion and distension of the prolapsed organs.

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[*] Wisconsin Alumni Research Foundation

MISCELLANEOUS DISEASES

Nordskog and Clark (1945) farrowed a bit early (101 to 111 days, average 107), they noticed 'no cases of typical abortion ' Distinguishing between 'early farrowing" and abortion might be difficult

DIAGNOSIS

Dysgalactia and diseases causing weak and stillborn pigs in the sow can be differentiated from ergotism by adequate udder development and response of some forms of dysgalactia to injections of pituitary hormone solutions Farmers will often remark about the apparent lack of physiological preparation for farrowing by the

sow when poisoned by ergot The gangrenous effects of ergotism may be differentiated from those of swine erysipelas by the dry gangrene nature of ergotism The preliminary swelling of the ears noted in erysipelas is lacking in ergotism

TREATMENT

It is doubtful if the diagnosis will be made before symptoms are well developed At that time removal from the source of ergot will be the only practical treatment. Symptomatic treatment of gangrenous portions is indicated

REFERENCES

Burns, P W 1953 Veterinary toxicology classroom notes Texas A & M College, College Station Texas

Burnside, J E., Sippel, W L Forgacs, J Carll, W T, Atwood, M B and Doll, E R 1957 A disease of swine and cattle caused by eating moldy corn II Experimental production with pure cultures of molds Amer Jour Vet Res 18 817

Carll, W T., Forfacs, J., Herring A S and Maillandt, B G 1955 Toxicity of *Aspergillus fumigatus* substrates to animals Vet. Med 50 210

Case, A A 1956 Personal communication

Christensen J J., and Kernkamp, H C H 1936 Univ Minn Agr Exp Sta Tech Bull No 115

Emmel, M W., and Sanders D A 1942 Univ Fla Press Bull 574

Forgacs, J., and Carll, W T 1955 Preliminary mycotoxic studies on hemorrhagic disease in poultry Vet Med 50 172

————, Herring, A S and Maillandt, B G 1954 A toxic *Aspergillus clavatus* isolated from feed pellets Amer Jour Hyg 60 15

Fröhner E., and Völker, R 1950 Lehrbuch der Toxikologie fur Tierartze, 6th ed Ferdinand Enke Stuttgart

Gadjusek, D C 1953 Acute infectious hemorrhagic fevers and mycotoxicoses in the Union of Soviet Socialists Republics. Med Sci Publ 2, Walter Reed Army Medical Center Washington, D C.

Glasser, K., Hutka, E., and Wetzel, R 1950 Die Krankheiten des Schweines 5th ed M & H Schaper, Hannover

McErlean B A 1952. Vulvovaginitis of swine Vet Rec. 61 559

McNutt S H., Purwin, P., and Murray C 1928 Vulvovaginitis in swine Jour Amer Vet Med Assn 73 181

Mitchill, F E., Hale, M W., and Cox, D H 1956 Tifton Diagnostic Lab Notes Ga Vet 8 (No 6) 12

Nordskog, A W., and Clark, R T 1945 Ergotism in pregnant sows female rats and guinea pigs Amer Jour Vet Res 6 107

Pullar, E M., and Lerew, W M 1937 Australian Vet Jour 13 58 Quoted by McErlean

Sippel, W L. Burnside, J E., and Atwood M B 1953 A disease of swine and cattle caused by eating moldy corn Proc 90th Ann Meet Amer Vet Med Assn, p 174

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Metritis, Mastitis, and Agalactia

Metritis

Metritis, or inflammation and infection of the uterus, often occurs following farrowing, dystocia, or abortion Metritis is part of the agalactia syndrome so frequently encountered as a clinical entity in swine practice

ETIOLOGY

Metritis is usually the result of infection of the genital tract during farrowing and possibly at the time of service to an infected boar Streptococci or *Escherichia coli* have both been isolated in pure culture from uterine swabs from sows showing puerperal infection and agalactia The physical stress associated with farrowing, uterine fatigue, and atony of the uterine musculature, retention of shreds of placenta, or a retained fetus is conducive to uterine infection and inflammation

CLINICAL SIGNS

Gilts and sows having puerperal uterine infection show inappetence and depression They will be found lying in their beds, shivering or trembling The temperature ranges from 103° to 107° F The udder is hot and congested and milk flow is inhibited This is believed to be one of the primary causes of agalactia in sows The signs of the disease appear in 1 to 3 days following farrowing A copious whitish to yellowish discharge from the vulva is seen by the end of the first or second day

Metritis resulting from retained fetuses, capillary thrombosis, pressure necrosis or laceration, and infection following dystocia is accompanied by a more watery serosanguineous, foul smelling discharge from the vulva Fever, inappetence, and agalactia will be present

PATHOLOGICAL CHANGES

The necropsy findings will vary considerably with causative factors Sows that have died as a result of dystocia or the retention of one or more fetuses will be found to have the abdominal cavity filled with reddish, foul smelling transudate The uterine horns will be flaccid and will not be involuted The uterine walls show a bluish discoloration and are very friable Capillary thrombosis is extensive The horns of the uterus will contain a foul smelling reddish exudate, shreds of fetal membranes, and an emphysematous fetus or fetuses

DIAGNOSIS

The disease is diagnosed by the history of recent farrowing and the clinical signs observed such as inappetence, agalactia, and a white mucopurulent discharge from the vulva

Manual exploration of the vagina and body of the uterus may reveal a retained

granulomatous areas which gradually en large until the section appears to be a tumorous mass several inches in diameter

Postparturient fever and caked, con gested, edematous udder are discussed un der the agalactia syndrome (page 516)

Gilts and sows suffering with acute post parturient gangrenous mastitis of the coli form type, have an extreme toxemia They lie in the bed, very depressed The tem perature may be subnormal to 107° F, de pending upon the stage of the disease at the time of the first examination The skin over the rear udder sections is purple the sections swollen and edematous, and the secretions serosanguineous The skin discoloration, with various degrees of sloughing, may extend over most of the udder The mortality is very high Some cases respond if treatment is instituted early The litter will die due to starvation unless it is separated from the sow and fed on sow s milk substitute Sows recover ing from the disease will have indurated udder sections and should be slaughtered as they are poor risks for future nursing litters

PATHOLOGICAL CHANGES

During the initial stage of subacute mas titis the skin, stroma, and parenchymatous tissues are involved in an inflammatory re action The usual lesions of inflammation are present in the glandular area There is edema and leukocytic infiltration into the glandular area The ducts leading from the alveoli are filled with inflammatory products

Depending on the degree of inflamma tion and the amount of capillary throm bosis, the gland becomes infiltrated with connective tissue Many of these glands are atrophied and fibrosed

In glands infected with *Actinomyces bovis, Actinobacillus lignieresi,* or staphylo cocci a granulomatous process may develop These granulomatous masses contain areas of necrosis and walled-off abscesses In some cases an ulcerated discharging sinus may be present

Alder (1951) and Langham and Stock ton (1953) have very aptly described the lesions of coliform mastitis Langham and Stockton state

The lesions were confined primarily to the mam mary glands and the lymph nodes The former structures were greatly swollen and firm The skin covering the mammary glands showed a purplish discoloration On cut sections there were areas of congestion, hemorrhage and ne crosis The interlobular tissue was very edema tous The supramammary lymph nodes were greatly enlarged due to edema congestion and hemorrhage Microscopic sections of the mam mary glands reveal extensive changes in the epithelium of the acini characterized by vacu olar degeneration necrosis and desquamation In the lumina of the acini were some lym phocytes and polymorphonuclears desquamated epithelial cells and clumps of bacteria The stroma showed congestion of the capillaries and extensive edema A few of the blood vessels contained thrombi The supramammary lymph nodes had areas of congestion hemorrhage and edema The lymph sinuses contained large numbers of polymorphonuclears a fibrinous exudate and clumps of bacteria

DIAGNOSIS

Mastitis involving individual udder sec tions is diagnosed by keen visual observa tion and manual examination of the indi vidual udder sections A gland may appear swollen and the pig which had been nurs ing it found fighting the other pigs for a place to nurse On closer examination the secretions from the section will be found to be changed in character Some secretions are watery with a few flakes and in others the secretion is purulent Some of these glands will atrophy and never return to milk Other glands may become granu lomatous and enlarged The granulom tous sections will be especially apparent when the litter is weaned and the normal udder sections are atrophied

The diagnosis of acute postparturient gangrenous mastitis is rather apparent due to the toxemia, the swollen, discolored, purplish udder, and the serosanguineous secretion

TREATMENT

Many individual infected glands are never treated Occasionally a gland is in fused with penicillin and streptomycin If the sow is showing generalized signs and

fetus at the pelvic inlet. Shreds of placenta and a foul smelling brownish fluid may be encountered on the floor of the vagina or in the body of the uterus

TREATMENT

A combination of penicillin and strepto mycin intramuscularly to control the in fections is indicated Uterine tone and in volution should be stimulated by the ad ministration of 25 mg of stilbestrol and 2– 4 ml of posterior pituitary extract

A retained fetus can sometimes be ex tracted manually If it can not be extracted, 1 qt of mineral oil and ½ oz of soluble tetracycline pumped into the uterus will assist in keeping down an absorption and infection from the uterus and will facili tate the passage of fetal and placental shreds

PREVENTION

A survey of the premises should be made to determine the sanitary conditions and management practices In cases where it appears that the disease is a herd problem, the farrowing houses or central farrowing house should be thoroughly cleaned and steamed or chemically disinfected

The sows, gilts, and boars should be tested to eliminate genital infections such as brucellosis and leptospirosis which cause abortions or weak pigs and uterine infec tion

Unsanitary, septic lay assistance to par turition should be discontinued

Proper diet and exercise are very im portant Fat, under exercised sows will have a high incidence of dystocia, weak atonic uteri, and secondary metritis

Mastitis

Infectious mastitis occurs sporadically Occasionally sows will contract acute gan grenous mastitis due to coliform and staph lococcic organisms A puerperal Strep tococcus infection is prevalent in some large herds of swine Mastitis metritis, and agalactia are common to this syndrome Caking, congestion, and edema of the ud der, as well as agalactia may be seen as a result of improper diet and exercise but should not be confused with infectious mastitis

Chronic indurative and granulomatous mastitis involving one or more glandular sections is seen especially in older sows

ETIOLOGY

Merchant and Packer (1956) list the following organisms associated with masti tis in the sow

  1 Streptococci and staphylococci
  2 *Spherophorus necrophorus*
  3 *Actinomyces bovis*
  4 *Actinobacillus ligneresi*
  5 *Corynebacterium pyogenes*
  6 *Mycobacterium tuberculosis*

Staphylococci *Actinomyces bovis*, and *Actinobacillus ligneresi* have been isolated

from granulomatous udder sections

Adler (1951), Helmboldt (1953), and Langham and Stockton (1953) have iso lated the coliform organisms *Aerobacter aerogenes* from the mammary glands and spleen of sows dying of acute postparturi ent gangrenous mastitis

Udder injury from laceration by the sharp canine teeth of the suckling pig may inoculate the gland or adjacent tissue

CLINICAL SIGNS

A subacute or chronic streptococcic or staphylococcic mastitis will involve one or more udder sections There are very few systemic signs shown, as the infection ap pears to be confined to the affected gland The milk secretion is reduced or entirely absent from that section and the pig nurs ing it will be hungry and will rob nurse from another gland Many of these glands become atrophied, indurated, and fail to secrete at future farrowings The results of this type of mastitis are seen frequently in older sows

Udder sections which become infected with staphylococci, *Actinomyces bovis*, or *Actinobacillus ligneresi* often develop

postparturient fever and agalactia resembles clinically the so called beta hemolytic Streptococcus syndrome in whelping bitches He states that, as in the bitch, the sow shows no signs of the condition until immediately following parturition The symptoms include inappetence, agalactia and some degree of pyrexia Hackett reports recovery of streptococci from both the uterus and udder in this syndrome Jackson (1952) reports that he has isolated overwhelming numbers of *Escherichia coli* from the uterine discharges, the intestinal contents of the pigs, and from blood smears taken from the hearts of the pigs He associates *Escherichia coli* infection with parturient fever and agalactia

Agalactia will be present as one of the signs of any systemic disease such as hog cholera, erysipelas, swine influenza, and transmissible gastroenteritis

Sows that farrow in damp cold quarters may become chilled, their litters become chilled and fail to nurse Many of these sows develop agalactia

DIAGNOSIS

Diagnosis is usually very apparent The sow shows inappetence and depression Her litter is hungry and beginning to show weakness, depression, and various stages of hypoglycemia Frequently one or more pigs may have died before the veterinarian is called

A thorough history and physical examination should be made in order to be sure that some infectious disease will not be overlooked If there is evidence of a serosanguineous discharge or foul odor from the vulva, a manual vaginal palpation should be made At times a retained fetus or some portion of the placenta may be encountered at the pelvic inlet. Laceration of the vagina or pressure necrosis from the intervention by lay personnel may be diagnosed

The consistency of the feces should be examined

The udder should be palpated The secretion, if any, should be obtained, examined, and the color of the mammae observed (Purplish discoloration and serosanguineous secretion is indicative of acute mastitis)

Extremely high temperatures are frequently indicative of the clinical syndrome of agalactia, especially in very hot weather, though they may be associated with an infectious disease such as acute erysipelas and influenza

Previously normal sows which have just farrowed, and are now showing inappetence, depression, failure to let the pigs nurse, and have hungry depressed litters should strongly be suspected of having the clinical syndrome commonly diagnosed as agalactia

TREATMENT

The aim of treatment is to restore milk flow in as brief a time as possible Due to the fact that the exact cause for failure of milk secretion is not always known, a treatment covering as broad a range of therapeutic correction as possible is selected

Administration of 5 0 ml of posterior pituitary extract is suggested The oxytocic principle causes the secretion of milk within a few minutes in a high percentage of cases The smooth muscles of the uterus contract and, many times, considerable amounts of the detritus of placenta and other uterine inflammatory products are expelled Occasionally a retained fetus is expelled An antibiotic combination of penicillin and streptomycin is administered to control any puerperal infection

In cases of constipation the digestive tract is emptied by the administration of 2–3 ml of lentin given intravenously in the ear vein or subcutaneously behind the ear Many sows vomit a few minutes after administration of the lentin and soon afterward the bowels move In severe constipation, a high enema is administered, followed orally by 1 oz. of cascara sagrada or 1 oz of sodium hyposulphate.

One treatment will start milk secretion in many of the cases encountered The litter usually dies when sows continue to stay off food and lapse back into agalactia Some older pigs can be saved if given sup-

has a fever, penicillin and streptomycin or sulfonamides are administered Valuable purebred sows having granulomatous udder sections are sometimes treated by surgical removal of the affected gland Commercial sows are usually marketed when a large number of glands are affected or the granulomatous masses interfere with nursing the litter

Treating postparturient coliform mastitis is very discouraging The toxemia in many cases is so overwhelming that the sow dies, regardless of treatment If an

early diagnosis is made, streptomycin 5 mg per lb of body weight given at 8 hour intervals, is sometimes beneficial

PREVENTION

When mastitis of any type occurs more than sporadically on a farm, a survey of the premises and a study of the management should be made Sanitary conditions housing feeding, and management should be corrected, as is necessary in any profitable swine enterprise Sows with discharging glands should be isolated or marketed

Agalactia

Agalactia is a very prevalent syndrome seen in sows at farrowing time or during the nursing period It results in the death of many litters of pigs during the first few days following farrowing

Pigs which are 10 days of age can usually be saved by supplemental feeding of a sow s milk substitute

ETIOLOGY

There are many causes for agalactia Milk secretion is dependent on so many factors that it is often very difficult to establish the definite cause for agalactia in the clinical cases that come to the veterinarian s attention In sows recently farrowed or at other stages in the lactation period, factors such as diet extreme hot weather, environmental changes, nervousness, constipation, systemic disease, dystocia retained placenta metritis mastitis hormonal imbalance, and disease in the newborn suckling pigs may individually or in combination cause the clinical syndrome

CLINICAL SIGNS

Agalactia itself is really the sign of interference with some phase of physiological milk secretion The most prevalent type of clinical syndrome which we refer to as agalactia is encountered at farrowing or during the first 2 or 3 days following farrowing It will be apparent that the baby pigs are hungry in various stages of starvation and hypoglycemia Sows with

agalactia are uneasy and lie in the sternal position, up on the udder, and fail to roll over on the side so the gland will be exposed for nursing Some sows lie out flat and permit the pigs to suckle but fail to secrete milk The sow is partially or completely off feed, with a temperature ranging from normal to 106° F She is depressed and may not get up unless forced to do so The udder is firm and congested but the teats are flaccid On an attempt to hand milk, no milk or only a few drops can be squeezed from the teats The sow shows various degrees of trembling which is probably associated with chilling due to intoxication and fever If she is postparturient 24 hours or more, a copious milky mucoid white discharge from the vulva is often seen Bowel movements are absent or dry and scanty The udder is hot and congested

This type of agalactia appears to be due to a combination of overfeeding concentrated rations when the sow is penned for farrowing, and to the autointoxication associated with a sluggish digestive tract weak atonic uterus a detrition of shreds of placenta, and in many cases, secondary puerperal uterine infection This syndrome has been encountered in several sows on the same farm It appears that a puerperal uterine infection must be present in many cases showing this disease syndrome

Hogg (1952) states that this type of

REFERENCES

Metritis

ANTHONY, D J   1955   Diseases of the Pig 4th ed Baillière Tindall and Cox London, p 238
FREEMAN, T D   1955   Treatment of bovine and porcine metritis   Report of 10 cases Vet Med
       50 307

Mastitis

ADLER, H E   1951   Mastitis in sows associated with aerobacter infection No Amer Vet 32 96
ANTHONY, D J   1955   Diseases of the Pig 4th ed Baillière, Tindall and Cox London p 239
FALSTER, L B   1950   Acute mastitis in sows clinical data Mich St Coll Vet 10 114
HELMBOLDT, C F   1953   Coliform mastitis in swine Case report Vet Med 48 80
LANGHAM, R F, AND STOCKTON, J L   1953   Cases of aerobacter mastitis in a sow Mich St Coll
       Vet 13 112
MERCHANT, I A, AND PACKER, R A   1956   Veterinary Bacteriology and Virology 5th ed The
       Iowa State College Press, Ames Iowa

Agalactia

ANTHONY, D J   1955   Diseases of the Pig, 4th ed Baillière, Tindall and Cox London p 206
BEAT, V B   1956   So called milk fever in the sow No Amer Vet 37 276
BREEDS, F   1952   Common causes of agalactia in the sow Laboratory diagnosis and discussion
       Norden News 26 10
HACKETT, W C   Personal communication
HASTINGS C C   1955   Milk fever in sows No Amer Vet 36 102
HOGG A H   1952   Common causes of agalactia in the sow Vet Rec 61 191
JACKSON, B N   1952   Bacterial coli infection as a cause of agalactia in the sow Vet Rec 61 191
WELBOURN, W E   Personal communication

plemental feeding with a sow's milk substi-
tute

Symptomatic treatment is indicated for
the pigs in the litters of sows suffering from
agalactia Dextrose, 10 ml of a 5 per cent
solution, intraperitoneally, will correct the
hypoglycemia and give the pigs strength to
continue to nurse

Sows which completely dry up make a
gradual recovery Many can be rebred and,
with a change in feed and management,
will lactate normally for the next litter

PREVENTION

Recommendations for prevention should
include correction of management, diet,
exercise, and cleaning and disinfecting the
farrowing house or houses The possibility
of venereal infection should be kept in
mind If the boar can be incriminated in
transmitting genital disease, he should be
replaced

Gilts and sows should be kept on a good,
balanced ration high in alfalfa meal or on
legume pasture during the gestation period
They should be kept in good physical con-
dition but should not be allowed to be-
come too fat When they are penned for
farrowing the ration should be adjusted
by adding some bulky feed such as bran
and ground oats The usual amount of the
regular ration should be limited for a few
days before and after farrowing

Thyroprotein has been used, a few days
prior to and a few days after farrowing, in
the feed at the rate of 100 mg per pound
of feed Limited field trials have shown
some promise in temporarily stimulating
greater milk flow

It has been proved by many swine prac-
titioners that the proper use of a balanced
ration helps measurably in the prevention
of agalactia

The following rations have been used
successfully for several years in the Ohio
State University swine herds Four to five
hundred pigs per year are farrowed in this
herd and agalactia is rarely encountered

Sow Ration Hand-Fed, Winter

800 lb ground shelled corn
400 lb ground wheat or middlings
100 lb ground oats
70 lb meat scraps 50–55% crude protein
90 lb soybean oil meal 44% crude pro-
tein
200 lb dehydrated alfalfa meal
30 lb mineral ' Sacco V19
10 lb trace mineral salt
Vitamin D (1 million units per lb or 5
oz)

2,000 pounds and 5 ounces (Per cent crude
protein, 13 8)

Sow Ration Hand-Fed, on Pasture

900 lb ground shelled corn
400 lb ground wheat or middlings
500 lb ground oats
70 lb meat scraps
90 lb soybean oil
30 lb mineral Sacco
10 lb trace mineral salt

2 000 lb (Per cent protein, 13 06)

The above rations are fed at the rate of
4 to 6 pounds per day depending on the
size of the gilt or sow When the gilt or
sow is penned for farrowing, bran is sub-
stituted for about one half of the above
ration and continued for 2 or 3 days fol-
lowing farrowing

Sow Ration Free Choice

Welbourn has experienced good results
in preventing agalactia and in increasing
numbers and vigor in litters by starting
the following ration, free choice, about 10
days prior to breeding and carrying it
right through the farrowing period

600 lb corn
600 lb alfalfa meal
600 lb ground oats
200 lb pig and sow supplement 35% pro-
tein

2 000 lb (Per cent protein 12)

A mineral mixture should be supplied
free choice, with the above ration

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Hypoglycemia in Baby Pigs

Mann and Magath (1922) demonstrated that hepatectomy in the dog causes fatal hypoglycemia This brilliant observation has been confirmed many times The danger from hypoglycemia, even to life it self, has been well stated by Krehl (1955) He said 'The mammalian organism can not function without a constant supply of carbohydrate Any reduction of blood glu cose below a critical level will lead to dis aster, especially for the delicate tissues represented in the central nervous system which can use only glucose as a source of energy'

The significance of Krehl's statement is aptly illustrated by the syndrome of acute hypoglycemia in the baby pig This may be spontaneous or be induced experi mentally by fasting or by the administra tion of insulin [1] (Fig 37 1)

This discussion is concerned primarily with spontaneous or clinical hypoglycemia in the baby pig first reported by Graham et al (1941) These observations were concerned with severe hypoglycemia en countered in entire litters of newborn pigs on numerous Illinois farms For want of a better name at that time the syndrome was referred to as so called baby pig disease However, in the first as in subsequent re ports emphasis was directed to the intense hypoglycemia which characterized the dis

order Attempts to demonstrate the pres ence of an infectious agent in the blood tissues and gastrointestinal content of typi cally affected pigs were unsuccessful No characteristic gross pathological lesions were observed The stomachs of these pigs were usually empty, but in some a vari able amount of curdled milk was present

Sampson and associates in 1942 re ported that a syndrome indistinguishable from spontaneous or clinical hypoglycemia could be induced experimentally in healthy baby pigs by subjecting them to a relatively brief period of fasting 1 e 36 to 48 hours Subsequently, Hanawalt and Sampson (1947a, b) found that whereas newborn pigs were particularly susceptible to fasting or starvation hypoglycemia this was not true for pigs of weaning age Weanling pigs could be fasted or starved at 60-70° F for as long as 30 days without a dangerous fall in blood sugar (water and salt were allowed) Morrill (1952) showed that baby pigs fasted at 60° F developed fatal hypoglycemia in about 24 hours whereas death occurred in approximately 72 hours at 90° F

Goodwin (1955) at the School of Vet erinary Medicine of the University of Cam bridge, confirmed the Illinois observations On the basis of the latter findings and the results of his own observations, Goodwin concluded

(1) Following the reduction of the high con centration of liver glycogen found at birth and

---

[1] Mann and co workers showed that hepatectomy also causes fatal hypoglycemia in the pig

before the establishment of satisfactory gluco-neogenesis, a period of 3 to 4 days would occur in which the blood glucose concentration would be extremely unstable, fluctuating widely with the milk intake (2) Under the conditions of 1 above, this peculiarity of carbohydrate me tabolism could influence most of the morbidity of this period, directing a great variety of aetiological factors to be expressed in common symptoms and rendering fatal several con ditions that could be surmounted in later life by the physiology of the adult

ECONOMIC IMPORTANCE

Evidence obtained from two widely sepa rated swine producing areas, by Sampson and associates in Illinois and by Goodwin in England, suggested that spontaneous or clinical hypoglycemia is an important factor in deaths of baby pigs Since partial or complete agalactia is known to be com mon among sows after farrowing, it is safe to assume that many litters of pigs die from hypoglycemia caused by starvation Many of these pigs are probably crushed by the sow when they become weak or are in hypoglycemic coma Goodwin observed starvation hypoglycemia in his field studies He said that entire litters of pigs in a pedi gree herd of Large White swine were dying on the second day of life

The time of death suggested complete starva tion from birth and clinical examination of the litters confirmed the hypoglycemic syn drome Furthermore, the stomachs of the dead pigs were ballooned and together with the lower bowel, completely devoid of milk Bac terial cultures from piglets were negative for pathogenic organisms The sows exhibited few clinical signs (not even loss of appetite after farrowing) apart from complete agalactia Under these conditions in the field the hypo glycemic syndrome is seen in its most acute form and without complications

Similar observations were made in Illi nois

Although agalactia of varying intensity is probably the chief cause of hypoglycemia in baby pigs, cases do occur that presum ably are not due to a lack of milk secretion In these cases, as mentioned earlier, the stomach of the pig contains a variable amount of solid curd Goodwin also called attention to such cases It is not always clear whether the syndrome is then caused

by (1) some abnormality of the colostral milk, (2) a failure of normal digestion and absorption, or (3) other obscure influences Goodwin describes this group of cases under hypoglycemia secondary to some other clinical disorder and points out that death of the pig may or may not result from hypoglycemia Thus, hypoglycemia is often a primary condition and probably results from either complete starvation or from a ' progressive reduction in milk in take, or secondary to some other clinical disturbance If primary, death usually re sults from hypoglycemia, if secondary, death, if it should occur, may or may not result from the effects of low blood sugar

ETIOLOGY

In the last analysis, a failure of gluconeo genesis would seem to be the most signifi cant influence in the pathogenesis of fatal hypoglycemia of the baby pig Neverthe less, agalactia must be looked upon as a frequent predisposing or indirect cause It is not uncommon to find a number of sows in a herd that show either partial or complete agalactia (Carroll and Krider 1956) No satisfactory explanation can be given at this time for the failure of so many sows to secrete milk Adequate nutrition during pregnancy is essential for lactation, but agalactia in many instances does not appear to be associated with improper nutrition because usually a majority of sows in affected herds provide enough milk for their litters

Mastitis, metritis, and other disorders are probably responsible in some cases, but these would seem to be in the minority Perhaps most sows affected with agalactia simply do not possess an inherent capacity to secrete a good flow of milk The report by Goodwin lends support to this view He stated that in one herd, agalactia after farrowing affected so many sows that the entire breeding stock had to be replaced Thus, agalactia should prove to be an in teresting problem for more research

Besides agalactia, other important pre disposing causes of hypoglycemia would appear to involve (1) disturbances in the
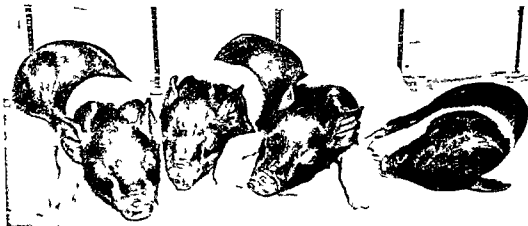
FIG 37 1—Hypoglycemia in baby pigs Upper, severe spontaneous hypoglycemia Middle, hypoglycemic coma produced by fasting Lower, hypoglycemic coma produced by insulin injection Increasing stupor and finally coma are manifested as the blood sugar level continues to fall below 40 mg per 100 ml Convulsions may or may not be observed in hypoglycemia of the pig Note that in the middle figure the four control, or non fasted, pigs show no signs of hypoglycemia The normal range of the blood sugar level of newborn pigs is about 60–140 mg per 100 ml with an average of approximately 100 mg (From Dukes, The Physiology of Domestic Animals, 7th ed Courtesy Comstock Publishing Associates Ithaca, N Y, 1955, p 539)

many respects to those observed in hypo glycemia induced by fasting or by the ad ministration of insulin (Sampson and Gra ham, 1943) The symptoms in the early stage of clinical hypoglycemia are often missed because weakness and lessened activ ity are not readily detected True blood sugar in vigorous, healthy baby pigs usually fluctuates between 60 and 140 mg per 100 ml with an average of approximately 100 mg (Sampson *et al*, 1942, Morrill 1946, Hanawalt and Sampson, 1947a, b Goodwin, 1955) So long as the concentra tion of sugar in the blood remains above 50 mg per cent, the pig may not show any unusual behavior As the sugar level falls below 40 mg per cent, however, definite symptoms become noticeable When the level reaches 20 mg per cent or less, con vulsions and coma are imminent and al ways characterize the terminal stage of the syndrome At this stage, the pig is unable to stand and often manifests galloping and other movements of the legs The head may be pulled backward or twisted to one side A decrease in body temperature ac companies the fall in blood sugar and is often lower than 95° F in the terminal stage The skin is cold and the hair often stands upright Heart sounds become faint and imperceptible, and the heart rate is slowed. A weak squeal is sometimes emitted Most of these symptoms, as well as others, are also mentioned in Goodwin's excellent description of experimental fast ing hypoglycemia of the baby pig He de scribes these signs as follows

The newborn pig (once removed from the birth process and dry) is active, vigorous and strong on its feet, instantly grunting and in search of food unless replete and purposeful in behaviour The skin is reddish pink (when unpigmented) and very warm such a pig usually shows relatively little change in ap pearance until the blood glucose concentration has declined to about 50 mg per 100 ml, but thereafter its activity and indeed its whole metabolism appears to run down in clock work' fashion and in step with the blood glu cose The progressive reduction in metabolism is evidenced most readily by this steady de crease in activity at first the gait is uncertain and this deteriorates until the pig may maintain balance only with the additional support pro

vided by its nose on the ground or by resting on the carpi and straddling the hind legs Later it may rest on its abdomen, but ulti mately it falls on its side and shows no further activity until the onset of convulsions The latter are a common feature and although they may be incomplete (hesitant frequently in terrupted or reduced in extent) they are usually unmistakable The pig exhibits strong and regular galloping movements of the forelegs with full extension during each cycle Cor responding movements in the hind limbs are generally less extensive the legs moving in a half flexed position or being drawn up for ward against the abdomen During these move ments the head is drawn back slightly and the lower jaw champs rhythmically (typical air hunger signs) developing a froth about the mouth The period just preceding the first convulsion together with the convulsion peri ods themselves may be associated with a tor tuous rigidity of the trunk and neck Further signs of the declining metabolism are the fall in temperature (which can be appreciated by feeling the skin), increasing bradycardia (the heart rate can decrease from about 220 to 80 per minute or less between birth and the con vulsive period) and the associated circulatory changes such as skin pallor Pigs in such straits even when the blood glucose concentration registered as low as 7 mg per 100 ml have been restored to normality following prompt glucose therapy

PATHOLOGICAL CHANGES

As stated previously, no characteristic gross pathological changes are ordinarily observed at necropsy of pigs that die from uncomplicated hypoglycemia In some pigs the liver and kidneys are dark and con gested, but this can be explained by poor circulation during the terminal stage Mor rill (1916) found some lipoidal changes in the liver of many pigs affected with spon taneous hypoglycemia A few pigs with either spontaneous or experimental fasting hypoglycemia also showed urates in the kidneys, but apparently there was no ob struction or kidney dysfunction Morrill stated that the lipoidal changes in the liver might suggest complicating factors.

If a complicating condition such as scouring is involved in baby pig hypogly cemia a part or all of the gastrointestinal tract may be inflamed and injected The intestinal content is often foul smelling semiliquid, and either grayish or yellow

baby pig characterized by impaired appetite and abnormal digestion and absorption as may occur in scours or (2) other pathological conditions associated with the neonatal period (Fig. 37.2).

Evidence in support of a failure of gluconeogenesis is found in the observation that while healthy, vigorous baby pigs have from 2 to 6 per cent or more of liver glycogen, pigs that die from hypoglycemia

have only a trace of hepatic glycogen present (Sampson *et al.*, 1942; Morrill, 1946, Goodwin, 1955). Apparently the physiological mechanism of gluconeogenesis requires a number of days after birth of the pig before it begins to function effectively.

CLINICAL SIGNS AND DIAGNOSIS

Signs of spontaneous hypoglycemia are not pathognomonic but are similar in

Disease in the Newborn Pig
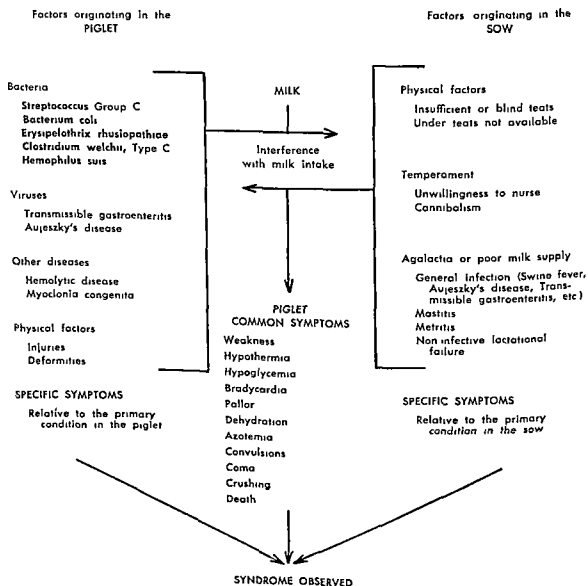
FIG. 37.2 — Scheme indicating some of the probable interrelations of physiological and pathological factors in deaths of baby pigs. (From Goodwin. Courtesy British Veterinary Journal, 1955, 3:361–72.)

KERNKAMP, H C H   1950 Differentiating physical characteristics of hypoglycemia uremia toxemia, and transmissible gastroenteritis in baby pigs No Amer Vet 31 729

KREHL W A   1955 The nutritional significance of the carbohydrates Borden's Rev Nutr Res 16 85

MANN, F C   1944 Studies on the dehepatized animal A review Jour Mt Sinai Hosp 11 1

———— AND MAGATH, T B   1922 Studies on the physiology of the liver 11 The effect of the removal of the liver on the blood sugar level Arch Int Med 30 73

MORRILL, C C   1946 A physiopathological study of the newborn pig with special reference to hypoglycemia Abst of Ph D Thesis Univ of Illinois

———— 1952 Studies on baby pig mortality X Influence of environmental temperature on fasting newborn pigs Amer Jour Vet Res 13 322

NUTRITION NEWS BULL. 1956 Nutritional disorders of baby pigs Ralston Purina Nutr News Bull 12, No 4

SAMPSON, J 1950 Hypoglycemia in baby pigs Vet Med 45 187

————, AND GRAHAM, R 1943 Studies on baby pig mortality III A note on experimental insulin hypoglycemia in the pig Jour Amer Vet Med Assn 102 176

————, HESTER, H R , AND GRAHAM R 1942 Studies on baby pig mortality II Further observations on acute hypoglycemia in newly born pigs (so called baby pig disease) Jour Amer Vet Med Assn 100 33

ish white in color and sometimes blood stained Other changes may be present, depending upon the nature of the primary disturbance

Kernkamp (1950) has described the comparative pathology and differential diagnosis of some common diseases of baby pigs, including hypoglycemia

TREATMENT

Mann and associates (1944) found that an injection of glucose was the most effective way to alleviate hypoglycemia in the hepatized animals Injections of sugar can be given either intravenously or intraperitoneally In the baby pig, the intraperitoneal route is preferable To be effective, however, glucose must be given early in the stage of the hypoglycemic syndrome Temporary stimulation and relief can be obtained when an injection is given after prolonged coma of one or more hours, but recovery is not the rule even though no complicating disease or condition is involved This is also true when glucose is injected to alleviate the effects of experimental hypoglycemia in the pig (Sampson and Graham, 1943, Sampson, 1950, Goodwin, 1955)

In hypoglycemia secondary to some other disorder, a favorable response is more certain if the first injection of glucose is given in the early stages of convulsions or coma the final outcome depends upon the nature of the primary disturbance In many of these cases, death occurs despite treatment in any form

If sterile glucose solution is used it can be given in 5 to 50 per cent concentration One or 2 ml of 50 per cent, or approximately 15 ml of 5 per cent glucose can be injected several hours apart. Some veterinarians prefer the 5 per cent solution because this concentration is near isotonic strength If the pigs can swallow, a procedure that has been recommended is to give each pig one tablespoonful of 50 per cent glucose solution three times daily The administration of sugar (or syrup) can be discontinued as soon as the pigs will drink milk or consume a milk substitute It is believed by some that scouring is less likely to occur when a good, balanced milk replacer is fed than when cow s milk is given either alone or as a part of a formula for orphan pigs (Nutrition News Bull , 1956) Overfeeding should be avoided The problem is simplified if the litter can be divided among several sows that give enough milk

PREVENTION AND CONTROL

Since the baby pig has difficulty maintaining a safe blood sugar level for approximately one to two weeks after birth if fasted or starved for even a relatively short time, especially in a cold environment, it is imperative that protection against such hazard be provided during this interval Prevention against chilling is important under any circumstance but particularly so when the pig obtains insufficient amounts of milk

A program which embraces all basic principles of good swine husbandry, viz proper nutrition of the sow during pregnancy, recognition of the urgent need of the litter for adequate nourishment during the first week or two after farrowing, and reasonable protection of baby pigs against exposure to chilling, infection, and other harmful influences should prove helpful in the prevention of losses from hypoglycemia

REFERENCES

CARROLL, W E., AND KRIDER, J L. 1956 Swine Production 2nd ed McGraw Hill Book Company Inc, New York

GOODWIN, R F W., 1955 Some common factors in the pathology of the new born pig Brit. Vet Jour 111 361

GRAHAM, R., SAMPSON J., AND HESTER, H R 1941 1 Acute hypoglycemia in newly born pigs (so-called baby pig disease) Proc. Soc Exper Biol and Med 47 358

HANAWALT, V M., AND SAMPSON, J 1947a Studies on baby pig mortality IV Chemistry of the blood during fasting and refeeding of weanling pigs Amer Jour Vet Res 8 73

——— AND ——— 1947b Studies on baby pig mortality V Relationship between age and time of onset of acute hypoglycemia in fasting newborn p gs Amer Jour Vet Res 8 35

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CHAPTER 38

Myoclonia Congenita

Some of the more common names which have been applied to the disease of baby pigs characterized by a congenital tremor are shakes, trembles, shivers, jumpy pig disease, and dancing pigs (Hughes and Hinman, 1936, Kinsley, 1922, Knilans, 1936, Lamont et al, 1950, Luke and Gordon, 1955, Nissley, 1932, Payen and Four nier, 1934) Kernkamp (1950) suggested that this disease be called *myoclonia con genita*

Continents on which the disease has been reported are North America, Europe, and Australia The distribution within the United States is only sketchily known, but its occurrence is apparently not limited to any one portion of the country No esti mates of the incidence of myoclonia con genita have been published, but the situ ation in the state of Minnesota can be cited as an example Over a period of ap proximately 18 months there were reports of over 150 litters of baby pigs affected with tremor These reports involved 35 different herds which were fairly evenly distributed throughout the swine raising areas of the state The significance of these numbers in terms of total incidence is not known Re ports from other countries indicate that the disease is fairly prevalent in some areas and may be increasing out of pro portion to the total increase in swine popu lation

The disease is known to occur in a wide variety of breeds and crossbreeds and so far there has come to light no information on breeds of pigs that are not susceptible to myoclonia congenita Although the over all mortality appears to be low, the neonatal death loss in individual litters may be relatively high

ETIOLOGY

The etiology of myoclonia congenita of baby pigs has not been demonstrated Early theories which postulated a genetic defect have been fairly well refuted by reports of closer observations Hindmarsh (1937) and Larsson (1955) reported that two matings of a specific boar to the same female produced one normal litter and one litter with tremors

Several authors, Brooksbank (1955) Florio et al (1956), and Hindmarsh, have put forth the suggestion that myoclonia congenita may be a viral disease Brooks bank postulates that a subclinical viral in fection in the dam during the gestation period may be the underlying cause No experimental evidence has been supplied to support these theories

Although the mechanism of action is not clear, outbreaks of myoclonia con genita often appear more or less simul taneously in several herds located in a given area In these instances it is usually found to be true that the same boar has been used to sire all of the affected litters

ewly acquired boar Although at present
t is impossible to say what role the boar
plays in the transmission of congenital
remor, it appears to be an important one
The disease seems to run its course in the
herd in one or rarely two farrowings

Nevertheless it may be safer in most cases
to replace the boar as soon as possible

Attempts by Larsson to reproduce myo
clonia congenita by mating pigs which have
suffered from the disease have not been
successful

REFERENCES

BROOKSBANK N H 1955 Trembles in p glets Vet Rec 67 576
FLORIO R FLACHAT Ch COTTEREAU Ph FLOCHON G TEDIDA M AND SAINT CYR R   1956
   Sur la Maladie des tremblements du porcelet Rev méd vet 57 209
HINDMARSH W L 1937 Trembling in young pigs Australian Vet Jour 13 219
HUGHES E H AND HINMAN R 1936 Trembling in pigs Jour Amer Vet Med Assn 89 96
KERNKAMP H C H 1950 Myoclonia congenita a disease of newborn pigs Vet Med 45 189
KINSLEY, A T 1922 Dancing pigs? Vet Med 17 123
KNILANS A J 1936 Trembling in pigs Jour Amer Vet Med Assn 89 590
LAMONT H G LUKE D AND GORDON W A M 1950 No 3—Some p g diseases Vet Rec
   62 737
LARSSON E K 1955 Om skaksjuka hos smagrisar Svenska Svinavelsforeningens Tidskr 9 119
LUKE D AND GORDON W A M 1950 Observations on some pig diseases Vet Rec 62 149
NISSLEY C M 1932 Shivers in pigs Jour Amer Vet Med Assn 81 551
PAYEN, B AND FOURNIER P 1934 Porcelets trembleurs Rec. méd vét 110 81
STROMBERG, M W 1956 Observations on a nervous condition of baby pigs Anat Rec 124 113
TURBES C RICHARDS A B AND ABREU B E 1956 Changes in the cerebellum in newborn pigs
   showing tremor Anat Rec 124 376

CLINICAL SIGNS

Animals afflicted with this disease almost invariably show the signs immediately or within a few hours after being born. The manifestation is essentially a tremor, with occasionally an associated hind limb weakness. The tremor may vary from a fine, almost imperceptible tremor to a coarse twitching of the limbs. This twitching may be so severe that the baby pig literally jumps off the ground with one or both hind limbs. The tremor may involve different skeletal muscle groups in varying degrees. Therefore some animals may show a marked head tremor, some a marked hind limb tremor, and some a more or less generalized tremor. In affected animals there is usually a dramatic cessation of tremor activity as soon as the pig lies down. The rhythmic, abnormal, muscle activity may cease entirely or be replaced by occasional twitching of single muscles or muscle groups. When the pig arises the tremor returns. Seriously affected animals show a continuous tremor while standing. As recovery progresses the tremor may become intermittent.

Several factors are known to aggravate the tremor. These are excitement, cold environment, ingestion of cold liquids, and parenteral administration of epinephrine (Stromberg, 1956).

Mild cases of tremor may cease to show any signs in a matter of hours. In others the tremor may persist for several weeks or months. Affected animals which no longer show tremor signs under normal conditions may begin to tremble under conditions of excitement or stress. The severity of symptoms during the first few days of life is not necessarily a criterion for predicting the time which will elapse before complete recovery.

In most cases of myoclonia congenita the prognosis appears to be good if the pigs survive the first 4 or 5 days after birth.

PATHOLOGICAL CHANGES

Turbes *et al* (1956) report finding pigment infiltration in the cerebellum of pigs affected with congenital tremor. The same authors have also noted degeneration of Purkinje's cells in some areas of the cerebellar cortex in affected pigs. These lesions cannot be said to be pathognomonic for this disease, however, and these findings have not been confirmed elsewhere.

Florio *et al* have noted what they felt to be thyroid abnormalities, but the majority of authors have failed to find either gross or microscopic lesions in pigs with congenital tremor.

DIAGNOSIS

Because of the unique nature of this disease, differential diagnosis is not especially difficult. Affected pigs usually appear healthy and normal except for the presence of tremor. Symptoms are present at birth or very shortly thereafter. The disease may affect part or all of a litter and the severity may vary within the litter. Spontaneous recovery may occur rapidly over a period of days or may be prolonged for several weeks.

TREATMENT

Several methods of treatment have been tried but none have been definitely proven to hasten the recovery of pigs showing the tremor. Since cold is known to aggravate the tremor, it follows that at least in cold surroundings the removal of these pigs to a warm environment may offer partial relief.

IMMUNITY

Since no infectious agent has been demonstrated as a cause of this disease, there is little which can be said with respect to immunity. Apparently females which have produced litters with tremor will usually produce normal litters at subsequent farrowings.

EPIZOOTIOLOGY AND CONTROL

In herds where myoclonia congenita is a problem the history usually reveals that the disease appeared in the offspring of a

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CHAPTER 39

Infectious Atrophic Rhinitis

Infectious atrophic rhinitis is a transmissible disease of swine characterized by atrophy of the nasal turbinates The atrophy is usually limited to the nasal turbinates but in severe cases rarefication of the nasal, premaxillary, or maxillary bones may occur The inferior scroll of the ventral turbinate is the region most commonly affected, although any portion of the dorsal, ventral, or ethmoid turbinates may be involved Lesions of this condition are most common in swine two to five months of age All diseases of swine causing turbinate atrophy are called infectious atrophic rhinitis As additional information becomes available, it may be desirable to reserve the term infectious atrophic rhinitis for the disease produced by one etiological agent Synonyms for this condition are *Schnuffelkrankheit, snovle syge,* sneezing sickness, sniffling disease dystrophic rhinitis, atrophic rhinitis, A R, and rhinitis chronica atrophicans

HISTORY

Franque (1830) published the first report describing this condition after its recognition in Germany He reported that affected swine did not fatten, developed an atrophy of the nasal and ethmoid turbinates, and in severe cases, a malformation of the nose The condition may have been present in Germany a considerable time prior to this report since Schneider (1878) was able to obtain evidence that the condition had been recognized for at least 70 to 80 years York (1911) reported typical clinical evidence of this condition in a herd of swine in the United States How ever the first group of workers to report that infectious atrophic rhinitis existed in the United States was Doyle *et al* (1944)

Etiology. Franque (1830) Hering (1842), Besnoit (1903), and Busolt (1912) believed that the lesions observed in this syndrome were suggestive of a nutritional deficiency Several of these workers reported that dietary supplementation helped correct the condition Bone meal, lime phosphate, and cod liver oil were mentioned as having beneficial results Schell (1890), Hintze (1909), Wirth (1910), and Ingier (1913) confused the condition with osteosarcoma and osteodystrophia fibrosa of the nasal cavity The possible role of in heritance accounting for the lesions has been mentioned by Franque (1830), Schneider (1878), Hoflund (1937a, b), Krage (1937), and Bottcher (1941) Franque (1830) and Radtke (1938) observed that short nosed swine tended to have more severe turbinate atrophy On the other hand, MacNabb (1948b), Gen dreau (1948), Gilman (1949), and Flatla and Braend (1953) failed to find any cor relation between facial conformation or degree of inbreeding and severity of turbi nate atrophy present

Several workers have confused acute bac terial rhinitis with infectious atrophic

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Several workers have confused acute bac terial rhinitis with infectious atrophic

[529]

agents capable of producing turbinate atrophy in swine Shuman et al (1963) found that swine influenza did not appear to be involved in the production of turbinate atrophy in a herd they studied Kernkamp (1952) felt that pulmonary and gastrointestinal disturbances were not especially characteristic of this condition Done (1955) reported the occurrence of inclusion bodies in the nucleus of certain cells of the tubuloalveolar glands of the nasal mucosa of young pigs affected with a rhinitis He reported that the inclusion bodies were present in the early stages of naturally occurring and experimentally induced infectious atrophic rhinitis

Several groups of workers have found that chronic bacterial rhinitis will produce turbinate atrophy Thus Gilman (1919) cited McKay's unpublished experiments as indicating that Spherophorus necrophorus and Pasteurella multocida acted synergistically to produce turbinate atrophy Gwatkin et al (1953) found that certain strains of P multocida alone would produce turbinate atrophy Gwatkin and Dzems (1953) extended these observations and found that certain P multocida from pneumonic swine lungs or atrophic turbinates would produce atrophic turbinates in pigs and in rabbits Flatla and Braend (1953) and Braend and Flatla (1954) reported that cultures of P multocida recovered from atrophic turbinates would produce typical turbinate atrophy when instilled intranasally in young pigs

McKay and Carter (1953b) reported that turbinate atrophy in swine could be produced by the instillation of rabbit abscess material produced by the injection of crude atrophic swine turbinate material subcutaneously into rabbits After one or two rabbit passages the rabbit abscess material consistently yielded Pasteurella multocida and L type colonies of Spherophorus necrophorus However, pure cultures of P multocida failed to produce turbinate atrophy in any of the inoculated pigs Schofield and Robertson (1954) noted that various isolates of P multocida failed to produce turbinate atrophy but

that this same organism did produce turbinate atrophy in one trial when inoculated concurrently with Pseudomonas aeruginosa Young pigs placed in a pen vacated 3 weeks previously by pigs with turbinate atrophy did not develop turbinate atrophy

Gwatkin et al (1951) again demonstrated that cultures of P multocida would produce turbinate atrophy when inoculated into suitable baby pigs These pigs were found to transmit the condition to contact pigs Gwatkin and Dzems (1953) amplified their previous findings that P multocida cultures were capable of producing turbinate atrophy in swine and rabbits The rabbit material was infectious for swine Switzer (1956) found that certain P multocida cultures isolated from atrophic turbinates would produce turbinate atrophy in baby pigs Heddleston et al (1954) were unable to correlate P multocida with turbinate atrophy in the pigs they examined and Switzer (1954b) clearly demonstrated that P multocida was not the cause of turbinate atrophy in an infected experimental herd

Borgmann (1953) demonstrated that the cases of turbinate atrophy he examined were not due to Erysipelothrix rhusiopathiae, as suggested by Messmore (1952 b)

Switzer (1956) reported that Haemophilus sp (very similar if not identical with H fecalis) recovered from atrophic swine turbinates produced turbinate atrophy when instilled intranasally in baby pigs He also noted that prolonged chemical irritation of the nasal cavities of experimental pigs frequently produced rhinitis with turbinate atrophy

A filter passing agent believed to be a pleuropneumonia like organism was isolated by Switzer (1954a) from the nasal cavities of swine with turbinate atrophy When Switzer (1953b) inoculated this organism intranasally into young pigs a gross turbinate atrophy occurred although a mild rhinitis with hyperplasia of the submucosal lymph nodes was noted The intraperitoneal introduction of this organism into young swine produced a severe

rhinitis Thus Imminger (1890), Koske (1906) Manninger (1930), and Eber and Meyn (1934) believed that acute rhinitis due to *Bacillus pyocyaneus* or *Pasteurella* sp was similar to infectious atrophic rhinitis

Many workers have expressed the opinion that this condition resembled a chronic infectious disease Franque (1830), Jensen (1916), Petersen (1925), Petersen (1926), Jensen (1933), Thunberg (1937), Radtke (1938), Thunberg and Carlstrom (1940), Reinboth (1940), Doyle *et al* (1944), Isa (1944), Connell (1945), McClelland (1945), Phillips (1946), Slagsvold (1946), Duthie (1947), and Moynihan (1947) believed that they were dealing with an infectious disease Although several of these workers attempted to transmit the condition experimentally, none reported success except Radtke (1938) The basis for successful transmission of the condition was developed by Jones (1947), Phillips *et al* (1948), and MacNabb (1948a) They found that pigs inoculated with atrophic turbinate material during the first few days of life frequently developed turbinate atrophy Jones (1947) and Gwatkin *et al* (1949) reported that pigs exposed at a few weeks of age did not develop lesions while pigs exposed very early in life did develop them

Smith (1953) observed that pigs 4 to 8 weeks of age did not develop lesions when placed in contact with infected swine Braend and Flatla (1954) noted that pigs exposed at 4 weeks of age developed mild turbinate atrophy while pigs exposed at 6 weeks of age did not On the other hand, Gendreau (1918) observed that pigs 7 to 8 weeks old acquired infectious atrophic rhinitis when placed in a pen which had previously contained infected pigs Doyle (1950) also observed that 10 week old pigs developed clinical evidence of the disease after introduction into an infected herd

Switzer (1951) reported that *Trichomonas* sp occurred in about 80 per cent of swine nasal cavities exhibiting turbinate atrophy and in only 2 8 per cent of grossly normal swine nasal cavities He was unable

to establish cultures of the trichomonad in the nasal cavities of experimental pigs and concluded that a mild rhinitis would probably favor its establishment He did succeed in establishing both the swine nasal trichomonad and *Trichomonas suis* in the bovine vagina Simms (1952) suggested that the nasal trichomonad was probably involved in one way or another in the production of turbinate atrophy Shuman *et al* (1953) noted that 40 7 per cent of a series of swine with turbinate atrophy harbored trichomonads in their nasal cavities while 15 6 per cent of the nonaffected pigs had trichomonads Spindler *et al* (1953) presented evidence indicating an etiological relationship between trichomonads and turbinate atrophy Ray (1953) concluded there was insufficient evidence to establish trichomonads as the etiology of infectious atrophic rhinitis Levine *et al* (1954) were unable to produce turbinate lesions in young pigs by the intranasal inoculation of bacteria free cultures of nasal trichomonads recovered from field cases of infectious atrophic rhinitis Brion and Cottereau (1954) noted that trichomonads were frequently present in the nasal cavities of swine with turbinate atrophy Hansen and Flatla (1955) reported that pigs with atrophic turbinates frequently harbored nasal trichomonads but that cultures of the trichomonad did not produce turbinate damage

Several workers have reported that a filter passing agent or agents would produce turbinate atrophy Radtke (1938), Phillips (1946), and Switzer (1953a and 1956) have observed on occasion that certain filtrates would produce turbinate atrophy Jones (1947), MacNabb (1948a), Gwatkin *et al* (1949), Schofield and Jones (1950), Gwatkin *et al* (1951), Flatla and Braend (1953), Braend and Flatla (1954), and Gwatkin *et al* (1954) found that filtrates of atrophic turbinates did not reproduce the condition It has been suggested by Radtke (1938), Reinboth (1910), Slagsvold (1946), Sandstedt (1918), and Switzer (1954b, 1956) that certain pneumonic swine lungs contain an agent or

kittens guinea pigs and rabbits failed to produce any gross lesions Gwatkin and Plummer (1949) were unable to produce rhinitis by the nasal instillation of crude material and filtrates from affected pigs into mature and baby mice hamsters guinea pigs and rabbits Shuman et al (1956a) noted that a turbinate atrophy factor was able to survive for about 3 weeks in the nasal cavity of the albino rat These workers also cited Schofield s report that the domestic cat could be a carrier of this condition

Switzer (1954d) noted severe turbinate atrophy in a few months old calf with a persistent nasal discharge *Streptococcus* sp was recovered from the nasal cavity of this specimen

Lesions Franque (1830) reported that swine affected with this condition developed an atrophy of the nasal and ethmoid turbinates with a subsequent mal formation of the nose and in severe cases exhibited nasal hemorrhage Spinola (1858) Haubner (1873) Schneider (1878) Schell (1890) Imminger (1890) Besnoit (1903) Koske (1906) Hintze (1909), Wirth (1910) Busolt (1912) and Ingier (1913) reported lesions they observed in cases of infectious atrophic rhinitis However during this period there had developed a tendency to refer to al most any involvement of the nasal cavity by this term so a diversity of lesions was recorded Jensen (1916) reviewed the literature that had accumulated about this condition and pointed out that 3 distinct disease syndromes were included under one name (1) acute infectious nasal catarrh (2) bone malformations such as osteopetrosis osteomalacia rickets or ostitis fibrosa deformans of the facial bones (3) a condition similar to the original one de scribed by Franque and characterized by a chronic atrophy of the nasal turbinates followed by a chronic purulent nasal catarrh

Petersen (1926) noted that the atrophic turbinates contained very little osseous tis sue and that atrophy of the turbinates was a permanent change Although Hollund

(1937?) believed the condition was a her editary defect he noted that if only *one* side of the nasal cavity was affected the nose was distorted laterally but that if *both* sides were affected the pig usually had a shortened nose He demonstrated turbinate atrophy by radiographs

Radtke (1938) concluded that the prin cipal lesions observed in this condition resulted from an inflammation of the nasal and sinus cavities The inflammatory re action appeared to involve the mucous membranes initially with subsequent action on the periosteum This resulted in alter ation of the osseous structures of the nasal cavity In cases where this process was more intense on one side lateral deviation of the nasal cavity was observed The normal for mation of the sinuses appeared to be de pendent upon proper function of the mucous membrane When the inflamma tory process involved the mucous mem brane of the sinuses there frequently was an alteration in the normal development of the sinuses especially the frontal sinuses with a resultant alteration in the contour of the skull Radtke concluded that the deformities of the skull observed in this condition resulted from the faulty develop ment of the nasal and ethmoid turbinates and the sinus cavities due to a chronic in flammation

Doyle et al (1944) noted that the prin cipal lesions were bone distortion atrophy and chronic inflammation of the nasal mucosa with some necrosis The charac teristic gross lesions were described by Phillips (1946) as due to a progressive dissolution of the softer bony structures of the nasal cavity A chronic inflammation of the nasal mucosa preceded the decalci fication of the nasal and ethmoid turbi nates While decalcification of the turbi nates was occurring a similar but less noticeable alteration of the harder facial bones occurred This produced distortion of the nose

The initial reaction observed by Schofeld (1949) to occur in the nasal cavity of swine affected with this condition was an infiltration of large lymphocytes and

fibrinous pericarditis, pleuritis, and peri tonitis An arthritis occurred in some cases The organism was recovered from field cases exhibiting similar lesions Carter and McKay (1953) reported that they believed many of the L type *Spherophorus necro phorus* colonies that McKay and Carter (1953a, b) recovered from the nasal cavi ties of swine and from rabbit abscesses pro duced by the subcutaneous inoculation of atrophic turbinate suspensions, were in reality pleuropneumonia like organisms Recovery of the previously reported organ ism from 20 of 28 pneumonic swine lungs was reported by Switzer (1954a) Carter (1954) found that baby pigs developed no lesions when inoculated intranasally with cultures of a swine pleuropneumonia like organism He isolated a pleuropneumonia like organism from 3 outbreaks of sero fibrinous pericarditis, pleuritis and peri tonitis in swine Young pigs inoculated intraperitoneally with these cultures de veloped lesions similar to those observed in the field case, and the organism was re covered from these lesions Switzer (1954b) reported the cultivation in artificial medium of the organism he had previously isolated A more complete description of this organism was presented by Switzer (1955), who proposed the name *Myco plasma hyorhinis* This organism was re covered from about 60 per cent of the swine nasal cavities he examined regardless of the presence or absence of turbinate atrophy Carter and Schroeder (1955, 1956) reported that a pleuropneumonia like organism was common in pneumonic swine lungs but did not appear to be the primary cause of the pneumonia Gwatkin et al (1954) found that 3 of 14 baby pigs inoculated with cultures of a swine pleuro pneumonia like organism had pus in their noses but no turbinate atrophy, and the remainder appeared normal

Switzer (1956) reported that nasal turbinate atrophy occurred in suitable ex perimental pigs as the result of intranasal instillation of either *P multocida*, *Alcali genes* sp, filter passing agent (s), or mild chemical irritants, indicating that all turbi nate atrophy in swine was not the result of a single etiological agent

Species affected. Most of the workers dealing with infectious atrophic rhinitis of swine have confined their observations to swine Therefore, it is to be expected that no mention is made of the presence or absence of similar lesions in other species Jensen (1916) stated that the con dition was confined to swine He noted that turbinate atrophy had not been ob served in any other species except man and that transmission from swine to man did not occur

Gwatkin et al (1953) found that rhinitis and turbinate atrophy were produced in some rabbits inoculated intranasally with cultures of *P multocida* and with crude turbinate suspensions from pigs with turbi nate atrophy They demonstrated serial passage of *P multocida* in the inoculated rabbits Gwatkin et al (1954) noted that after 8 rabbit nasal cavity passages, mate rial that had originally been very active in the production of swine turbinate atrophy produced only a moderate degree of turbinate atrophy in swine The *P multocida* recovered from this eighth pas sage material did not produce atrophy of swine turbinates Gwatkin and Dzenis (1955) reported that a suspension of nasal curettings from a pig with turbinate atro phy produced atrophic rhinitis in 6 of 7 rabbits All 7 yielded *P multocida* on cul ture The same material failed to produce atrophic changes in the nasal turbinates of white mice, white rats, or guinea pigs In two trials atrophic rhinitis was produced in rabbits for 3 and 14 passages, respec tively *P multocida* was isolated from a high percentage of these rabbit lesions

Thunberg and Carlstrom (1940) re ported they had observed several cases where cats and even dogs kept in contact with infected pigs developed a rhinitis and purulent conjunctivitis Jones (1947) ob served that cats kept with infected herds often developed a purulent rhinitis and occasionally a conjunctivitis However, inoculation of crude atrophic turbinate suspensions intranasally into 6 week-old

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ETIOLOGY

The etiology of turbinate atrophy of
swine has not been completely elucidated
However, there recently has been an encouraging
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It has been demonstrated repeatedly by
several different groups of workers studying
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Pasteurella multocida will produce typical
turbinate atrophy It is not possible to recover
this organism from all cases There
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certain localities *P multocida* constitutes
the primary etiological agent producing
of this is that in other herds in other areas
P multocida does not appear to play any
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The possible role in this condition of other
domestic or wild animals as reservoirs of
P multocida has not been adequately
studied although it has been rather conclusively
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that *P multocida* isolated from
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It has been demonstrated that in some
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Still another bacterium has been found
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This is an *Alcaligenes* sp that is very
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The fact that atrophy of the nasal turbinates
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It must be kept in mind that in addition
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In the United States, infectious atrophic rhinitis occurs in all of the major swine producing areas. It is estimated that from 5 to 10 per cent of the slaughtered swine in these areas have turbinate atrophy. Bennett (1951) reported that of the pigs over 3 weeks of age submitted to the Iowa Veterinary Medical Diagnostic Laboratory during a 6 week period, 59 had gross lesions of infectious atrophic rhinitis while 83 showed no nasal alteration. This was an incidence of 41.5 per cent.

Not all herds in a given area will have this condition, although it is probable that the majority of the herds will have a low incidence of lesions. On the other hand there will be some herds in which a high percentage of the individuals exhibit symptoms and lesions of this condition. It is these so called problem herds that have alarmed the swine industry.

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It has been demonstrated repeatedly by several different groups of workers studying this condition that certain isolates of Pasteurella multocida will produce typical turbinate atrophy. It is not possible to recover this organism from all cases. There are indications that in certain herds in certain localities P. multocida constitutes the primary etiological agent producing of this is that in other herds in other areas P. multocida does not appear to play any role in the production of turbinate atrophy.

The possible role in this condition of other domestic or wild animals as reservoirs of P. multocida has not been adequately studied although it has been rather conclusively shown that laboratory rabbits can function as reservoirs. It has been estab-

lished that P. multocida isolated from pneumonic swine lungs may also produce turbinate atrophy.

It has been demonstrated that in some herds a transmissible turbinate atrophy occurs that is not associated with P. multocida but is due to a large filter passing agent or agents that also occur in some pneumonic swine lungs. It has not yet been established whether this agent or agents play any role in the production of pneumonia. No information as to its field distribution or host range is available.

Still another bacterium has been found capable of producing turbinate atrophy. This is an Alcaligenes sp. that is very closely related to if not identical with A. fecalis. Again it is not known how widely distributed this organism is in the swine population. In certain cases this organism has been recovered from pneumonia occurring in association with turbinate atrophy. Pure cultures of the organism have produced pneumonia in experimental pigs. There appears to be a problem in maintaining the virulence of this organism in artificial medium so that it will produce turbinate atrophy and pneumonia. This organism may be relatively long lived. Rubber stoppered broth cultures have been found to be viable after storage for one year at 37° C.

The fact that atrophy of the nasal turbinates may not represent a specific disease is emphasized in that prolonged intranasal administration of chemical irritants to young pigs will produce rhinitis with some turbinate atrophy. It is quite likely that additional agents will be elucidated as the study of this disease syndrome progresses.

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the stroma of the submucosa The cells of the nasal epithelium appeared elongated or cuboidal and did not become stratified or squamous even in advanced cases Later there was an increase in the number of tubuloalveolar glands accompanied by a mild proliferation of the fibrous tissue elements of the stroma Proliferation of the osteoblasts was common However, the disappearance of the bony plates and trabeculae of the turbinates was considered to be the most outstanding characteristic of this condition

The observations of Schofield and Jones (1950) indicated the earliest gross changes of this condition were numerous small foci of congestion of the mucous membrane of the turbinate bones In severe cases the inorganic salts were almost completely removed from the turbinate bone in 2 to 4 weeks In many early cases the external surface of the nasal turbinate was practically free of any inflammatory exudate, but in more advanced cases a mucopurulent discharge was present The initial microscopic lesion consisted of scattered foci of degenerated and desquamated epithelial cells with cellular infiltration of the submucosa The infiltrating cells were mainly large lymphocytes that were not observed to extend beyond the outer layer of the periosteum even though the submucosa was densely packed with the cells They suggested that the portal of entry for the infection was the ducts of the tubuloalveolar glands is evidenced by the accumulation of neutrophils at this site Damage to the turbinate epithelium later became more extensive resulting in large denuded areas

In more advanced cases an increase in the number of tubuloalveolar glands was observed These were often distended with mucus to the extent that cysts were formed Even in the final stages the turbinate epithelial cells remained cuboidal or elongated and did not become stratified squamous epithelial cells as in primary atrophic rhinitis of man One of the earliest changes observed was proliferation of the osteoblasts In the areas of pro-

liferating osteoblasts there was frequently rarefaction of the bone In advanced cases the osteoblasts were present in enormous numbers and filled the space left by the disappearing bone This was regarded as an attempt to rebuild the bone The fibrous tissue elements of the stroma proliferated slowly, causing an increase in density and eventually surrounded both the arterioles and veins with a zone of dense fibrous tissue

Switzer (1956) reported that turbinate atrophy produced in experimental pigs by bacteria free filtrates and antibiotic treated crude inoculum usually had a minimum of surface exudate In experimental cases produced by *Alcaligenes* sp , *P multocida* or prolonged chemical irritation there was usually considerable mucopurulent exudate on the surface The chemical and bacterial materials appeared to produce irritation of the surface of the turbinate with resultant inflammatory reaction but had little visible effect on the osteoblasts It appeared that the reduced size of the turbinate resulted from its failure to grow at a normal rate The filter passing agent or agents appeared to produce little alteration of the epithelium but produced considerable infiltration of the submucosa with lymphocytes and lymphoblasts In some cases of turbinate atrophy produced by the filter passing agent or agents, the osteocytes and osteoblasts appeared to dedifferentiate into tissue resembling fibrous connective tissue In some cases this band of tissue replaced whole areas of the turbinate bone

DISTRIBUTION

The first recorded occurrence of infectious atrophic rhinitis was in Germany but it appears to occur in almost all parts of the world where there is an extensive swine industry The one exception seems to be England The condition was first reported from England in 1951 in the offspring of imported swine Rigid quarantine measures were believed to have eradicated the condition However Kernkamp (1956) observed that about 10 per cent of the slaughtered swine he examined on the

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The fact that atrophy of the nasal turbinates may not represent a specific disease is emphasized in that prolonged intranasal administration of chemical irritants to young pigs will produce rhinitis with some turbinate atrophy It is quite likely that additional agents will be elucidated as the study of this disease syndrome progresses

It must be kept in mind that, in addition to these primary agents, secondary bacteria and protozoa may increase the severity of turbinate atrophy We have not progressed sufficiently so that the effects of species interaction are evaluated as is important in any condition has been evaluated It never seems unlikely that *M septicum* and *M hyorhinis*, present in association with *P multocida*, may increase the effects Here further work is needed

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Others have suggested that the atrophic turbinates allow bacteria to gain access to the lungs where they intensify pre existing pneumonic lesions (3) Still other workers feel that some agents that produce turbi nate atrophy also produce pneumonia The etiology of turbinate atrophy is not ade quately defined to allow firm conclusions to be reached on this matter, although the evidence suggests that all three of these explanations may be correct under certain conditions

Herds of swine with a high incidence of turbinate atrophy and pneumonia are fre quently unthrifty Herds with a moderate to low incidence of turbinate atrophy and pneumonia usually make satisfactory gains Most workers dealing with turbinate atro phy believe that it has a mild retarding effect on the rate of gain of an affected animal but that it certainly is not the devastating condition it was reported to be soon after its recognition in this country Shuman and Earl (1956) have suggested that there is about a 5 per cent retardation of growth rate due to turbinate atrophy It is very apparent that several authors have tended to attribute all unthriftiness in a herd of pigs to turbinate atrophy when in reality the summation of several disease conditions was responsible for the unthrifty state of the pigs

PATHOLOGICAL CHANGES

The gross lesions observed in swine af fected with turbinate atrophy are confined to the nasal cavity and adjacent structures As is implied by the name, the most characteristic lesion is atrophy of the nasal turbinates The inferior scroll of the ventral turbinate is by far the most common site of atrophy being involved in the majority of the cases However, occasional specimens are encountered in which the atrophy is confined to the ethmoid or even the dorsal turbinate If the diameter of the nasal cavity is adequately decreased or if there is sufficient lateral distortion of the nasal cavity, the nasal septum will show some degree of buckling and may even have its dorsal or ventral attachments distorted laterally

A considerable amount of mucopurulent to caseous exudate is usually present on the mucous membrane of the nasal cavity The amount and character of the exudate depends to a considerable extent upon the age of the lesion and upon the secondary invaders that have become established In the more acute cases flecks of desquamated epithelium are present in the exudate The mucous membrane of the nasal cavity is usually somewhat blanched in appearance and gives the impression of being slightly edematous The mucous membrane of the sinuses especially of the frontal sinuses may be moderately hyperemic In a few cases a considerable amount of mucopuru lent exudate is present in the frontal sinuses

Although the nasal turbinates may be so atrophic that all that remains of them are small folds of mucous membrane attached to the lateral walls of the nasal cavity by far the most common finding is for the inferior scroll of the ventral turbinate to be from 20 to 50 per cent missing An al teration of the ventral turbinate that is observed very infrequently, but which is easily confused with turbinate atrophy, is a deep fold that is especially pronounced posteriorly When this occurs, the lateral attachment of the turbinate usually slants downward and the superior scroll is in creased in size This alteration appears to be a congenital defect

While the gross lesions observed in field cases of turbinate atrophy produced by different agents are quite similar, the micro scopic lesions are somewhat different The changes observed in turbinate atrophy that result from a chronic bacterial rhinitis usually consist of variable degrees of de squamation of the epithelium associated with cellular infiltration of the submucosa and hyperplasia of the tubuloalveolar glands The epithelium exhibits areas of erosion and partial desquamation and may even contain small cysts The number of goblet cells is increased A stratified

rium pyogenes, and *Brucella bronchisep tica* should be investigated for possible influence on the severity of the lesions present

CLINICAL SIGNS

The first signs noted in baby pigs affected with this condition are sneezing and sniffling These initial signs may be observed when the pigs are as young as one week The signs usually increase in severity but it must be cautioned that rhinitis in young pigs does not always indicate that turbinate atrophy will develop Some herds are observed in which acute sneezing and sniffling in the baby pigs subsides in a few weeks with no turbinate atrophy occurring The etiology of such cases of rhinitis has not been studied adequately Nonetheless, in the majority of herds in which the young pigs develop severe sneezing and sniffling turbinate atrophy subsequently develops

The sneezing, sniffling, and snorting observed in this condition results from the host's attempt to remove exudate from the nasal cavity Brisk exercise following a period of rest frequently produces an exacerbation of these symptoms There may be spillage of tears over the inner canthus of the eye with a resultant production of a moist crescent shaped area below the eye This moist area traps dirt and becomes black It is quite possible that the nasal opening of the lacrimal ducts is partially occluded with exudate in some cases but in other cases a mild conjunctivitis exists Thus, spillage of tears over the inner canthus of the eye may result from either failure of the lacrimal duct to carry away the secretions or increased lacrimal secretions stimulated by the conjunctivitis

A small quantity of clear to-purulent mucous exudate is often discharged from the external nares following sneezing As damage to the turbinate advances there may be flecks of blood in the exudate, and in some severe cases the trauma of sneezing will rupture some of the more exposed blood vessels and mild to profuse nasal hemorrhage occurs

The bones that form the nasal cavity and sinuses may be involved to some extent in this condition This is evidenced by their failure to grow at a normal rate When the damage is approximately equal on both sides of the nasal cavity, the length and diameter of the nasal cavity are reduced This is observed in the living animal as shortening of the nose The skin and subcutaneous tissue continue to develop at a normal rate and form wrinkles just posterior to the snout When the alteration is more severe on one side, the nasal cavity may be twisted toward the more severely affected side This lateral distortion may even progress so that the nasal cavity is twisted at a 45° angle Some people have placed considerable importance upon lateral distortion of the nasal cavity as a means of diagnosing turbinate atrophy This is unfortunate because the great majority of pigs with turbinate atrophy have no lateral distortion of the nose Admittedly in a few herds a considerable portion of the pigs do have twisted noses It is not yet possible to explain why a considerable number of the pigs in some herds evidence nasal distortion while in most herds they do not

When the frontal sinuses fail to develop at a normal rate due to the turbinate atrophy syndrome, there is reduced width between the eyes and an altered head profile The head profile tends toward that of a young pig instead of undergoing the alteration typical of maturity

In some herds affected with turbinate atrophy, sporadic cases of encephalitis occur This results from extension of bacterial infections through the damaged cribriform plate into the brain

It is very common to observe pneumonia concurrently present in a herd of swine affected with turbinate atrophy Three general explanations have been offered (1) Some believe that the damaged nasal turbinates allow foreign material access to the lung with resultant pneumonia (2)

FIG 39 5—Dorsal view of the head of a 3½ month old pig with lateral distortion of the nose

FIG 39 7—A view of the head of an 8 month old boar with severe turbinate atrophy The normal contour of the head has not developed The nose is somewhat shortened with wrinkling of the skin back of the snout

FIG 39 6—Cross section of the nasal cavity of the pig head shown above in Figure 39 5 There is almost complete atrophy of one ventral turbinate, a reduction in the diameter of the nasal cavity and distortion of the nasal septum

FIG 39 8—Cross section of the pig head shown above in Figure 39 7 There is almost complete atrophy of both the dorsal and ventral turbinates

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FIG 39 1—Dorsal v ew of the head of a normal
8 week old p g

FIG 39 3—Dorsal view of the head of an 8
week old p g with mild turb nate atrophy No
detectable alteration of the head has occurred

FIG 39 2—Cross sect on of same p g head shown
above in F gure 39 1 The nasal cav ty appears
normal

FIG 39 4—Cross section of same p g head shown
above n F gure 39 3 Note the m ld atrophy of
the nferior scroll of the ventral turb nate

body rhinitis occurs in England This condition is believed to result in turbinate atrophy under certain conditions Structures of epithelial origin are primarily involved in this condition The cells of the tubuloalveolar glands in certain areas develop a swollen nucleus containing conspicuous intranuclear inclusions Necrosis of the glands and ducts occurs and may develop into purulent foci Massive infiltration of the submucosa with lymphocytes occurs The surface epithelium is said sometimes to undergo metaplasia to a stratified squamous type The fact that there have been no reports of the occurrence of this inclusion body rhinitis in the United States has little significance since the matter has not yet been adequately investigated

No alteration of any of the osseous tissues other than of the nasal region, has

FIG 39 10—The same tissue section shown in Figure 39 9 Here, dedifferentiation of the osteocytes and osteoblasts is seen in greater detail Giemsa s stain X 420

been reported to occur in infectious atrophic rhinitis

DIAGNOSIS

At the present time it is not possible to diagnose turbinate atrophy in the living animal with sufficient accuracy to facilitate eradication of the condition However, the use of X ray and rhinoscopic examination has proved to be of interest and their value has been investigated by Earl and Shuman (1953) Shuman and Earl (1953), Braend and Flatla (1954), and Shuman and Earl (1955) It is axiomatic that any diagnostic procedure based upon observation of gross lesions cannot be utilized to detect lesion less carrier animals

If typical symptoms are present and lateral distortion of the osseous tissue of the nasal cavity is present it is reasonably certain that turbinate atrophy will be observed upon necropsy examination of the animal However, even the most experienced observers cannot positively ascertain that a specific live pig is completely free of turbinate atrophy

Most cases of turbinate atrophy are detected by examination of a cross section of the nasal cavity made at the level of the first premolar tooth This is the usual site of the maximum development of the scrolls of the nasal turbinates A power meat saw produces less distortion of the nasal turbinates than does a hand saw, although the latter can be used satisfactorily At this level the superior scroll of the ventral turbinate usually exhibits two complete turns while the inferior scroll exhibits one-and a quarter turns and in appearance is somewhat suggestive of a very blunt fish hook At this level the scrolls of the ventral turbinate appear to fill the major portion of the nasal cavity The ventral meatus is slightly larger than the medial meatus The nasal septum is normally straight in appearance It must be cautioned that sectioning of the nasal cavity anterior to this level will reveal a different development of the scrolls of the turbinates and may lead to an erroneous diagnosis of

cuboidal epithelium usually replaces the normal pseudostratified columnar ciliated epithelium as the germinal layer attempts to compensate for the desquamation Strati fied squamous epithelium has never been observed by the author to cover atrophic swine turbinates although it is reported to be common in cases of human turbinate atrophy Clumps of debris and bacteria may adhere to the damaged epithelial sur face

The cell types infiltrating the submucosa appear to be predominantly neutrophils and lymphocytes Some of the larger lymphocytes are probably lymphoblasts No tendency toward perivascular cuffing is ob served The small nodules of lymphoid tis sue normally present in the submucosa un dergo hyperplasia There does not appear to be any appreciable increase in the size or number of blood vessels in the sub mucosa although there does appear to be a slight thickening of the walls of the ex isting vessels It has been suggested that this is due to the contraction of the vessels as the total area of the vascular network is decreased, due to reduction of the size of the turbinate

The tubuloalveolar glands undergo hyperplasia The ducts of the glands may contain debris Osteoclasts are not present in significant numbers regardless of the cause of the turbinate atrophy Exami nation of tissue sections from some bacte rial turbinate atrophy cases discloses little alteration in either the appearance or number of osteoblasts In other cases there appear to be local areas of mild prolifer ation of the osteoblasts Examination of tissues from some of these cases of bacterial rhinitis creates the impression that a failure of normal growth of portions of the nasal turbinate due to the chronic rhinitis is an important consideration in accounting for the reduced size of the turbinate

A somewhat different type of lesion has been observed in experimental cases of turbinate atrophy produced by a large filter passing agent (s) There is only mild involvement of the epithelium The pri mary cell type present in the submucosal infiltration that occurs is the lymphocyte The tubuloalveolar glands undergo mild hyperplasia In some of the cases examined there has been very extensive proliferation of the osteoblasts This appears to be a dedifferentiation of the osteocyte and osteo blast into a more primitive type of tissue resembling fibrous connective tissue This type of tissue reaction is reviewed by Wil ton (1937)

It has been reported that an inclusion

FIG 39 9—A section of the inferior scroll of the ventral turbinate of an experimental pig This case of turbinate atrophy was produced by a large filter passing agent or agents The osteo blasts and osteocytes are dedifferentiating into tissue resembling fibrous connective tissue The epithelium has been torn from a portion of the tissue in preparation of the section Giemsa s stain X 100

This appears to work satisfactorily However, experimental evidence is not yet available to establish the length of time necessary to eliminate the etiological agents from contaminated lots and equipment The work that has been done suggests that a shorter period may be satisfactory

At the present time it appears that swine-to-swine transmission is the primary mode of spread of this condition, however, it must not be overlooked that under certain conditions other species may be carriers of an agent (especially *Pasteurella multocida*) that may cause turbinate atrophy in swine.

CONTROL

No satisfactory control or eradication measures are yet available that can be applied on an industry-wide basis. Any one of several procedures can be applied to an individual herd if the value of the animals warrants it. In any of these measures it is necessary that concurrent diseases and management abuses be eliminated. The simplest but least effective control measure is continual culling of visibly affected animals. This appears to reduce the degree of exposure in some herds to the point that few outward manifestations of turbinate atrophy are observed.

A second control plan is very similar to one suggested in England for the control of virus pneumonia of pigs Under this plan, bred females are housed in isolated lots and are never allowed contact with any other swine except their offspring until they are culled. The individual litters remain separate until a month after removal

of the sow at weaning time Breeding stock is selected from those litters that have evidenced no symptoms. A new herd is built up from this nucleus and is not allowed contact with any other swine Not all of the litters will be completely free of symptoms, but in those litters where transmission from the sow does occur the symptoms and lesions will be markedly reduced

A third control plan, used by Switzer (1954b, c), is to allow the sow to nurse the baby pigs and then remove them, when only a few hours of age, to an isolation area where they are reared by hand This has been modified by Johnson *et al* (1955) by removing the pigs at birth and then returning the pigs to the sow at intervals to be nursed The pigs are not allowed contact with the anterior portion of the sow As soon as the pigs have a fill of colostrum they are removed and reared by hand A method that completely bypasses this syndrome, as well as other respiratory disease is to procure the offspring by hysterotomy and raise them in as nearly sterile an environment as possible This procedure is discussed in Chapter 53 It has been shown that catching the pigs at birth on a sterile cloth, with subsequent removal to an isolated area, will also break the cycle of transmission. Shuman *et al.* (1956b) used this system to establish a herd free from infectious atrophic rhinitis. However, it must be cautioned that under farm conditions, the hand rearing of baby pigs that have received no colostrum is beset with many enteric disease complications and should be tried out on a small scale before being undertaken on a large scale.

REFERENCES

Bennett, P. C· 1951. Some angles on atrophic rhinitis. Proc U. S Livestock San Assn, p 201
Besnoit· 1903 Quelques considerations sur la "maladie du reniflement" du porc. Rev. vét. 28.397.
Borgmann, R : 1953. Infectious atrophic rhinitis unrelated to swine erysipelas. Vet. Med. 48 97, 101
Böttcher, H · 1911 Die Schnüffelkrankheit, ihre Ursachen und Verhütung Zeitschr f Schweine zucht, p. 251. (Original not available for examination; abstr. in Jahresb Vet. Med 69 119)
Braend, M, And Flatla, J. L : 1954 Rhinitis infectiosa atroficans hos gris Nord Vet. Med 6 81.
Brion, A, And Cottereau, P,. 1951 La rhinitis atrophique contagieuse du porc Rev med vet 105 705.
Busolt, K· 1912 Beitrage zur Kenntnis der Schnüffelkrankheit der Schweine. Inaug Diss Giessen, Germany).

turbinate atrophy The person conducting the examination must be familiar with the normal development of the nasal turbinates at the level examined At the present time no basis is available for the diagnosis of atrophic rhinitis except demonstration of turbinate atrophy

A more time consuming technique is to section the head longitudinally so as to split the nasal septum which is then dissected The lateral attachment of the ventral turbinate is severed with a pair of sharp scissors and the turbinate removed It can then be cross sectioned at various levels with sharp scissors for a critical appraisal This procedure has proved very helpful when material free of extraneous contamination is to be collected

TREATMENT

There are two general considerations that always should be evaluated before any treatment program is formulated for swine affected with turbinate atrophy The first of these concerns concurrent diseases The second concerns equipment and feeding and management practices It is uncommon to encounter a herd of pigs that are being reared under a good swine husbandry program and have only turbinate atrophy that ire not making reasonably satisfactory gains Direction of efforts toward elimination of concurrent diseases and management abuses usually gives better results than attempts to arrest the turbinate atrophy process

Relatively high levels of some of the broad spectrum antibiotics fed continuously to market pigs affected with turbinate atrophy have frequently been reported by field veterinarians to improve the rate of gain of the pigs It has usually been observed that reduction in the level of the antibiotic fed is followed by an exacerbation of respiratory symptoms

There are available commercial nasal instillations containing sulfonamides and other drugs that are advertised as having therapeutic effect against turbinate atrophy Until more is known about the etiology of this complex disease syndrome it is very difficult to evaluate the merits of such preparations

EPIZOOTIOLOGY

The primary mode of transmission of infectious rhinitis appears to be from pig to pig by means of infective aerosols Exposure may occur at any time in the life of the animal but turbinate atrophy usually develops only in those animals that are exposed at a few days or weeks of age Animals exposed later in life may develop mild cases of turbinate atrophy but usually exhibit symptoms of rhinitis which subside and which may leave the animal a carrier Repeated exposure of young pigs under conditions favoring aerosol transmission usually results in a high incidence of severe lesions Overcrowding of young pigs in damp quarters and subjecting them to frequent chilling supply some of these conditions

One of two general case histories is frequently associated with swine herds that have developed symptoms and lesions of turbinate atrophy sufficiently severe to alarm the owner One of these case histories is that the condition has existed at a low level for several years but has become progressively more severe This increase in severity may occur in one season or may span several farrowings Hutchings (1951) and Smiley (1953) have suggested that the disease takes about 3 years to build up to the point that it is a herd problem However it appears that this build up does not always occur even though this observation does apply in many cases The second general case history is that the owner had not observed this syndrome in his pigs until after the introduction of new breeding stock

In general turbinate atrophy does not appear to be transmitted by exposure to an environment that has been free of infected swine for a few days although a few exceptions to this are recorded in the literature It is a frequent practice to suggest to the swine owner that both lots and equipment should be given a 3-months rest prior to restocking with clean swine

Kerruish, D W 1956 Atrophic rhinitis in pigs other than Landrace Vet Rec 68 541

Koske, F 1906 Der *Bacillus pyocyaneus* als Erreger einer Rhinitis und Meningitis haemorrhagica bei Schweinen Arb a d k Gsndhtsamte 23 542

Krage, P 1937 Das Auftreten der Schnuffelkrankheit bei Schweinen in Ostpreussen und deren Bekampfung Deutsch tierarztl Wschr 45 129

Levine, N D, Marquardt W C and Beamer P D 1954 Failure of bacteria free trichomonas to cause atrophic rhinitis in young pigs Jour Amer Vet Med Assn 125 61

McClelland, S H 1915 Infectious rhinitis — bullnose of pigs West Farm Leader 10 357

McKay, K A, and Carter G R 1953a Some observations on the isolation cultivation and variation of *Spherophorus necrophorus* associated with infectious atrophic rhinitis, liver abscesses and necrotic enteritis Canad Jour Comp Med 17 299

——, and —— 1953b A preliminary note on the bacteriology and experimental production of infectious atrophic rhinitis of swine Vet Med 48 3o1 368

MacNabb, A L 1948a Rhinitis Rep Ontario Vet Coll p 12

—— 1948b Relationship between facial conformation and susceptibility to infectious rhinitis in swine Rep Ontario Vet Coll, p 64

Manninger, R 1930 Ansteckender Nasenkatarrh der Schweine Tierheilk u Tierzucht 7 350

Messmore, H L 1952a Erysipelas in swine No Amer Vet 33 308

—— 1952b Erysipelas in swine No Amer Vet 33 385

Moynihan, I W 1947 Rhinitis of swine II An effort to transmit chronic atrophic rhinitis of swine Canad Jour Comp Med 11 260

Petersen, A 1926 Infektios (enzootisk) naesekatarrh hos svinet og dens behandling Maanedsskr Dyrl 38 1

Petersen, G 1925 Fra praksis Maanedsskr Dyrl 37 241

Phillips, C E 1946 Infectious rhinitis in swine (bull nose) Canad Jour Comp Med 10 33

—— Longfield, H F and Miltimore J E 1948 Porcine infectious rhinitis experiments Canad Jour Comp Med 12 268

Radtke, G 1938 Untersuchungen uber die Ursache und das Wesen der Schnuffelkrankheit des Schweines Arch wiss prakt Tierheilk 72 371

Ray, J D Jan–Feb 1953 A practical discussion of swine diseases Haver Glover Messenger p 5

Reinroth, W W 1940 Ueber das Wesen und Bekampfung der Ferkelgrippe und der Schnuffelkrankheit der Schweine (Nach Beobachtungen aus der Praxis) Inaug Diss Leipzig

—— 1940 (original not available abstr in Deutsch tierarztl Wschr 49 200)

Sandstedt H 1948 De vanligaste svinsjukdomarna i Sverige och atgarder mot dem Norsk Vet Tidskr 60 355

Schell 1890 Osteoidsarcom in den Gesichtsknochen der Schweine (Schnuffelkrankheit) Berl Arch p 223 (original not available abstr in Jahresb Vet Med 10 76)

Schneider, A 1878 Ueber die sogenannte Schnuffelkrankheit der Schweine Deutsch Zeitschr f Thiermed u vergleich Path 4 183

Schofield, F W 1948 Pathology of atrophic rhinitis in swine Rep Ontario Vet Coll, p 138

—— and Jones T L 1950 The pathology and bacteriology of infectious atrophic rhinitis in swine Jour Amer Vet Med Assn 116 120

—— and Robertson, A 1953 Further studies in the pathology and bacteriology of infectious atrophic rhinitis of swine Proc. Book, Amer Vet Med Assn, p 155

Shuman, R D Andrews J S and Earl F L. 1956a Atrophic rhinitis in swine Yearbook of Agr U.S.D.A., p 350

——, and Earl F L 1953 Atrophic rhinitis III The evaluation of the rhinoscopic examination for its diagnosis Jour Amer Vet Med Assn 122 7

——, and —— 1955 Atrophic rhinitis V An effort to obtain atrophic rhinitis free pigs by selection and isolation Jour Amer Vet Med Assn 127 427

——, and —— 1956 Atrophic rhinitis VII A study on the economic effect in a swine herd Jour Amer Vet Med Assn 129 220

—— Shalkop, W T and Durbin, C G 1953 Atrophic rhinitis I A herd survey Jour Amer Vet Med Assn 122 1

—— and Stevenson, J W 1956b Atrophic rhinitis VI The establishment of an atrophic rhinitis free herd of hogs Jour Amer Vet Med Assn 128 189

Simms, B T 1952 Trichomonads associated with atrophic rhinitis of swine. Rep Chief U S Bur Anim Ind, Agr Res Admin p 70

Slagsvold L 1946 Smittsom hostesyke of nysesyke hos gris Norsk Vet Tidskr 58 445

Smiley, R S 1953 Infectious atrophic rhinitis in Ohio Vet Med 48 10

Smith H C 1953 Field cases of atrophic rhinitis Proc. Book, Amer Vet Med Assn p 118

Spindler, L A Shorb D A and Hill, C H 1953 The role of trichomonads in atrophic rhinitis of swine Jour Amer Vet Med Assn 122 151

Spinola, W T J 1858 Handbuch der speciellen Pathologie und Therap e für Thierärzte, 1st ed Vol 1 August Hirschwald Berlin, p 382

Switzer W P 1951 Atrophic rhinitis and trichomonads Vet Med 46 478

—— 1953a Studies on infectious atrophic rhinitis of swine I Isolation of a filterable agent from the nasal cavity of swine with infectious atrophic rhinitis. Jour Amer Vet Med Assn 123 45

CARTER G R. 1951 Observations on pleuropneumonia like organisms recovered from swine with infectious atrophic rhinitis and Glassers disease Canad Jour Comp Med 18 246

———— AND McKAY, K A 1953 A pleuropneumonia like organism associated with infectious atrophic rhinitis of swine Canad Jour Comp Med 17 413

———— AND SCHROEDER J D 1955 Pleuropneumonia like organism associated with pneumonia in swine Canad Jour Comp Med 19 219

————, AND ———— 1956 Virus pneumonia of pigs in Canada with special reference to the role of pleuropneumonialike organisms Cornell Vet 46 344

CONNELL, R 1945 A disease called 'bullnose' occurring in swine in Prairie Provinces Canad Jour Comp Med 9 221

DOVE, J T 1955 An inclusion body rhinitis of pigs (preliminary report) Vet Rec. 67 525

DOYLE, L P 1950 Rhinitis of swine Proc U S Livestock San Assn p 276

———— DONHAM C R, AND HUTCHINGS L M 1944 Report on a type of rhinitis in swine Jour Amer Vet Med Assn 105 132

DUTHIE R C 1947 Rhinitis of swine I Chronic atrophic rhinitis and congenital deformity of the skull Canad Jour Comp Med 11 250

EARL, F L, AND SHUMAN R D 1953 Atrophic rhinitis II The rhinoscopic examination of swine as a means of diagnosing atrophic rhinitis Jour Amer Vet Med Assn 122 5

EBER, A., AND MEYN, A 1931 Beitrag zur infektiosen Rhinitis (Schnüffelkrankheit) der Schweine Acta Path et Microbiol Scand 18 86

FLATLA J L. AND BRAEND M 1953 Infectious atrophic rhinitis in pigs Studies on the etiology Internat Vet Cong Proc. Part 1 p 180

FRANQUE 1830 Was ist die Schnüffelkrankheit der Schweine? Deutsch Zeitschr f gesam Tier heilk 1 75

GENDREAU L A 1948 Field observations on infectious swine rhinitis Canad Jour Comp Med 12 291

GILMAN J W P 1919 Inherited facial conformation and susceptibility to infectious atrophic rhinitis of swine Canad Jour Comp Med 13 266

GWATKIN, R. AND DZENIS L. 1953 Rhinitis of swine VIII Experiments with Pasteurella multo cida Canad Jour Comp Med 17 454

———— AND ———— 1955 Rhinitis of swine X Further experiments with laboratory animals Canad Jour Comp Med 19 139

————, AND BYRNE, J L. 1953 Rhinitis of swine VII Production of lesions in pigs and rabbits with a pure culture of Pasteurella multocida Canad Jour Comp Med 17 215

———— GREIG, A S, AND GRINEWITSCH, C 1954 Rhinitis of swine IX Further studies on aetiological agents Canad Jour Comp Med 18 341

————, AND PLUMMER P J G 1919 Rhinitis of swine IV Experiments on laboratory animals Canad Jour Comp Med 13 70

————, AND BYRNE J L. 1951 Rhinitis of swine V Further studies on the aetiology of infectious atrophic rhinitis Canad Jour Comp Med 15 32

———— AND WALKER R V L. 1919 Rhinitis of swine III Transmission to baby pigs. Canad Jour Comp Med 13 15

HANSEN, M A. AND FLATLA, J L. 1945 Trichomonader i neseslimen hos gris og deres relasjon til Rhinitis atroficans Nord Vet Med 7 660

HAEBNER, G K 1873 Schnüffelkrankheit der Schweine Die inneren und äussern Krankheiten der landwirtschaftlichen Hausfangethiere 6th ed P Parey Berlin Germany, p 202

HEDDLESTON K L, SHUMAN, R D, AND EARL F L 1951 Atrophic rhinitis IV Nasal examination for Pasteurella multocida in two herds affected with atrophic rhinitis Jour Amer Vet Med Assn 1 5 223

HERING E. 1842 Specielle Pathologie und Therapie für Thierärzte, 1st ed Ebner and Seubert, Stuttgart Germany p 139

HINTZ, R 1909 Das Wesen der Schnüffelkrankheit der Tiere Archiv wiss prakt Tierheilk 35 535

HOLLAND S 1937a Orientering over sjukdomen nyssjuka (rhinitis chronica atroficans) hos svin ur klinisk synpunkt Svensk Vet Tidskr 42 189

———— 1937b Nyssjukans etiologi Svensk Vet Tidskr 42 561

HUTCHINGS, L M May June 1951 Infectious diseases of pigs Norden News p 7 11

IMMINGER, 1840 Ein Beitrag zur infectiösen Rhinitis der Schweine (Schnüffelkrankheit) Wschr f Tierheilk p 1.5

INGLIS A 1913 Über die b e der Schnüffelkrankheit am Rumpf und Extremitätenskelett auf treten len Veränderungen Frankfurter Zeitschr f Path 12 270

IVA J M. Nov. 1941 Bullnose in pigs Country Guide p 16

JENSEN C. O. 1916 Om snoflesyge hos svinet Maanedskr Dyrl 28 277

———— 1933 Über die rhinitis chronica atrophicans des Schweines Acta Path et Microbiol Scand Supplementum 16 172

JOHNSON, F K., BONE, J F., AND OLDFIELD, J E. 1955 Atrophic rhinitis in swine 1 Methods of control in a purebred herd No. Amer Vet 36 191

JONES F L. 1917 Rhinitis in swine Agr Inst Rev - 271

KRANKAMP, H C H 1952 Infectious atrophic rhinitis No Amer Vet 33 68

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Necrotic Rhinitis and Exudative Epidermitis

Necrotic Rhinitis (Bull Nose)

The term *bull nose* is the very widely recognized name for a swine disease which has been observed for many years More technically it is also known as *necrotic rhinitis* The long time usage of the com mon terminology by both veterinarians and swine producers resulted in a tendency to refer to any diseased condition of the nose or snout of pigs as bull nose This led to considerable misunderstanding and confu sion when atrophic rhinitis became a widely publicized swine disease

Research work has not yet answered many questions regarding atrophic rhinitis and some of the misunderstanding and con fusion between the two diseases, bull nose and atrophic rhinitis, still exists Both diseases are observed most frequently in growing pigs In each, the name by which it is identified merely describes the con dition produced by the disease Both may be present at the same time in a single individual In atrophic rhinitis there is a gradual atrophy or disappearance of a portion of the bony and cartilaginous tis sues which make up the air passageway in the central portion of the nose In necrotic rhinitis, or bull nose, the development of the disease produces an abscess in the soft, fleshy tissue which surrounds the harder tissue forming the air passageway

True bull nose is of interest because it is one of the few swine diseases which is

becoming less common During the past several years the number of pigs produced and raised has gradually increased but fewer cases of bull nose are being observed Veterinary practitioners usually attribute this decreased incidence to improvement in management and production practices

ETIOLOGY

For many years the specific cause of necrotic rhinitis, or bull nose, has been thought to be the bacterial organism *Spherophorus necrophorus* Continued study of this organism and its effect in sev eral species of animals has created some doubts as to its primary significance as a causative agent of the necrotic processes in which it can be found It is quite common in nature, especially in environments of high animal populations and so is readily avail able to contaminate any wounds, abrasions, or other injuries of the mouth and snout areas of the pig The crushing of baby pig canine teeth with a pair of pliers can provide such a wound Poor and in adequate sanitary conditions are conducive to wound contamination by *S necroph orus*

In some instances the presence of *S ne crophorus* cannot be demonstrated by stain ing, cultural, or laboratory animal inocu lation methods Such failures, however, should not be accepted as conclusive proof the organism is, or was, not present Invari

Switzer W P 1953b Studies on infectious atrophic rhinitis of swine II Intraperitoneal and intranasal inoculation of young pigs with a filterable agent isolated from nasal mucosa of swine Vet Med 48 392

―――― 1951a A suspected PPLO in Iowa swine Iowa Vet 23 9

―――― 1951b Studies on atrophic rhinitis Proc Book Amer Vet Med Assn p 102

―――― 1951c Observations on infectious atrophic rhinitis Proc U S Livestock San Assn p 363

―――― 1951d Unpublished observation

―――― 1953 Studies on infectious atrophic rhinitis IV Characterization of a pleuropneumonia like organism isolated from the nasal cavities of swine Amer Jour Vet Res 16 510

―――― 1956 Infectious atrophic rhinitis V Concept that several agents may cause turbinate atrophy Amer Jour Vet Res 17 478

Thunberg E 1937 Bidrag till nyssjukans etiologi Svensk Vet Tdskr 42 360

―――― and Carlstrom B 1910 Om nyssjuka hos svin fran epizootisynpunkt Skand Vet Tdskr 30 711

Wilton A 1937 Tissue Reactions in Bone and Dentine, 1st ed Henry Kimptom London

Wirth D 1910 Beitrage zur Frage uber das Wesen der sogenannten Schnuffelkrankheit Oester Monatschr f Tierheilk 35 351

York W K 1911 A herd condition of swine characterized by persistent sneezing and nasal hemorrhage Fort Dodge Bio Chem Rev 12 18

of skin conditions, or to skin manifestations of more generalized diseases

Exudative epidermitis is recognized only in young pigs and has not been observed in other species It is probably quite widely distributed, although the numerous names which have been used and the scarcity of accurate information can be used to discount an extensive distribution It is somewhat irregular or cyclic in prevalence observations indicating that its incidence and distribution may increase during a period of a few consecutive years and then decrease sharply for a variable length of time

ETIOLOGY

The cause of greasy pig disease is unknown The causative agent or factor of many diseased conditions can be classified as either infectious genetic, allergic or nutritional, however, Jones (1956) in his study of the clinical features of this disease was unable to develop any significant evidence of a common factor which suggested that the causative agent might be found in some one of these classifications

CLINICAL SIGNS

First evidence of the disease may be observed at any time during the period when the pigs are between 1 and 4 weeks old It is most commonly seen between 12 and 24 days of age The morbidity may be as low as 10 per cent, but instances occur when 100 per cent of the pig crop may be affected Reports are rather common of entire litters being affected while other litters in very close contact remain apparently healthy

The mortality also varies greatly, ranging between 5 and 90 per cent The most commonly reported mortality approximates 20 to 25 per cent The early symptoms consist of a slight listlessness a dull appearance of the skin and hair and the appearance of small thin scales on the skin surface. Over a period of only 3 to 5 days these symptoms increase in severity and become much more prominent The listlessness becomes apathy, the skin be-

comes slightly swollen, and increased amounts of skin secretions can be observed During further progress of the disease dehydration and weight loss become apparent skin secretions continue and accumulate as skin incrustations and the pig presents a very dejected and unthrifty appearance These skin excretions produce a greasy and sticky condition which often causes neighboring hairs to stick together As the skin excretions accumulate and thicken the surface hardens and often cracks giving the skin a thickened and fissured appearance The accumulation often favors a rapid growth of bacteria on the skin skin necrosis and the development of obnoxious odors The crusts and scabs often become very prominent on the head back and around the tail head The bacteria most commonly recovered from the necrotic skin surface are *Micrococcus* and *Streptococcus* spp however it is quite possible that under some of the scabs *Spherophorus necrophorus* may become established

In the majority of cases which terminate fatally the entire course of the disease requires only 5 to 10 days A few severely affected individuals may live as long as 3 weeks No significant body temperature increase has been observed during the early stages of the disease and when increases have been noticed later they are thought to be associated with secondary complications Pruritus is not present in greasy pig disease

PATHOLOGICAL CHANGES

Necropsy shows a marked dehydration and emaciation Superficial lymph glands are usually swollen and edematous The stomach and small intestine usually contain no food material and the contents of the large intestine are much more pasty and dry than normal The kidneys usually contain a visible amount of white or orange yellow granular precipitate in the calyces and pelvis None of these gross pathological changes can be considered specific for greasy pig disease The most characteristic changes are those of the skin

ably other bacterial organisms will be found in the abscesses of bull nose in addition to *S necrophorus* Some of these organisms may be only saprophytic, but pathogenic organisms are also present The most common of these other pathogens be long in the Micrococcus, Streptococcus Corynebacterium, Proteus and Pseudomonas groups Occasionally, Alcaligenes may be found These pathogens other than *Spherophorus necrophorus* are usually the most predominant and abundant organisms to be found in the bull nose abscess

CLINICAL SIGNS AND PATHOLOGICAL CHANGES

The abscess of bull nose is basically similar to abscesses found in many other locations In many cases they develop to considerable size and their presence is easily observed In comparison atrophic rhinitis is limited to the disappearance of portions of tissues within the air passage way and in the majority of cases there is no external evidence of such disappearance

Although the original site of the necrotic process is in soft tissue, the development of the lesion sometimes involves the bone of the nose and face and considerable destruction of bone tissue may occur The extensive development of this necrotic process in either fleshy or bone tissues can result in interference with the ability of

the animal to consume its food This together with the toxic effect of the necrotic material results in a lowering of the general health status and any natural or artificially stimulated resistance to other diseases In such cases the growth rate becomes uneconomical and the pigs develop into rough appearing, unthrifty individuals Because of the uneconomical growth rate and the lowered level of resistance, bull nose abscesses should not be neglected

TREATMENT

Owners should be advised to have the abscesses treated as soon as they are detectable Some individual pigs may overcome the infection through natural processes, however, if the abscess continues to develop and is ignored, even surgical treatment may not produce satisfactory results

Since bull nose is simply a bacterial abscess, treatment either by surgery or drug therapy is comparable with that of other abscesses and ulcers If satisfactory results do not follow the use of the treatment first selected, a change to another drug may give the desired results In a few instances it may be desirable to have antibiotic sensitivity tests conducted on the organisms found in the abscess Prevention is much more effective and economical than treatment Good sanitation plus farm safety practices in eliminating as many injury hazards as possible will result in continued decreased incidence of this disease

Exudative Epidermitis (Greasy Pig Disease)

Greasy pig disease is the expressive name that is popularly used in the swine producing area of the Midwest to describe a skin disease of young pigs Jones (1956) reported on the clinical and pathological aspects of the condition He defines it as an acute generalized dermatitis involving the entire body surface of young swine, characterized by sudden onset and a short course marked by hyperhidrosis, excess sebaceous secretion, exfoliation, exudation and without pruritus, resulting in loss of skin function extreme dehydration rapid exhaustion usually terminating in death

His observations led him to identify it as *exudative epidermitis* of pigs

With only a few exceptions, skin diseases of pigs are very poorly understood, and reports of the obscure conditions have led to the use of names such as pustular dermatitis, necrotic dermatitis, infectious dermatitis, exfoliative dermatitis, exudative dermatitis, and eczema Such skin conditions are termed obscure because of the scarcity of information concerning them especially regarding their etiology, and it is very uncertain whether all the names listed refer to one disease, a multiplicity

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Paralysis and Lameness

Paralysis and lameness are rather common in swine. Locating the cause of these disabilities and applying effective remedial measures sometimes present an important problem. Locomotory disabilities are generally due to pathologic changes in the nervous system or in the skeleton. Occasionally lameness results from disease in the skeletal muscles or in the hoofs. Lameness is perhaps associated more frequently with arthritis than with any other condition. Anything that interferes with the animal's locomotion results in economic loss to the owner if the disability continues for any length of time.

ETIOLOGY

The etiology of locomotory disability in swine is extremely varied. That portion which is attributable to affection of the nervous system results from infection, nutritional deficiencies, mechanical injuries, and occasionally from toxins or poisons. The nutritional deficiencies which appear to be of most importance in this connection are lack of vitamin A and pantothenic acid. It is likely that heritable factors also cause some disabilities of nervous origin. When the disability is traceable to the skeleton the cause is nearly always infection or nutritional deficiency. Staphylococci and/or streptococci are commonly found in arthritic joints of young swine. In older animals, *Erysipelothrix rhusiopathiae* is often found in early stages of arthritis.

The occasional cases in which skeletal muscle lesions are responsible result from infection, mechanical injury and rarely, from tumors.

The occurrence in baby pigs of an arthritis characterized by a fibrinous inflammation of the serous membranes of the joints complicated with pleuritis, peritonitis and pericarditis has been described by workers both in the United States and abroad. The disease was recognized in Europe as early as 1910 (Glasser *et al*, 1944), but the etiology still does not appear to be definitely established. Under the name of Glasser's disease the disease is described by Hjarre and Wramby (1942), Gualandi (1955) and Knott (1956) as being caused by *Haemophilus suis*. Bierer (1956) described a similar condition believed to be associated with *Pasteurella multocida* but at the same time suggesting a possible virus involvement. Albert and Mendlowski (1956) reported on a serosal condition in swine which they believed to be of nonbacterial origin. It is quite possible that all of these workers were concerned with the same disease which we will continue to call Glasser's disease until more information is available.

Lameness originating in the hoof is sometimes the result of mechanical injury to the foot such as results from walking on rough, frozen ground. Exanthematous diseases such as vesicular exanthema may cause marked lameness because of foot

surface which were described in the section on clinical signs   Routine bacteriological examination of the heart, liver, spleen, and kidneys usually gives completely negative results

DIAGNOSIS

Since the cause of greasy pig disease is unknown   no diagnostic methods based on the causative agent are available   For practical purposes the diagnosis must be based on a consideration of history and gross pathological changes

At least one other skin disease in pigs has been frequently mistaken and confused with greasy pig disease   When research studies indicated that parakeratosis of swine was due to a mineral deficiency or imbalance, many owners and practitioners hoped that greasy pig disease was perhaps an early form of parakeratosis and would respond to the preventive measures that were found to be effective against parakeratosis   Unfortunately these hopes did not materialize   Such a marked difference in response to treatment for parakeratosis exists between pigs affected with these conditions that the lack of response following such therapy can be used as a diagnostic differentiation between the two diseases   Age of the affected pig is still another differential feature between these two diseases   Greasy pig disease usually develops within the first 3 weeks following farrowing while parakeratosis is not detectable until some later age

Due to the fact that greasy pig disease develops in young pigs there is a possibility that it may be confused with transmissible gastroenteritis   The presence or absence of typical diarrhea and skin lesions suffices to distinguish between these two diseases   Differentiation from skin parasitism and fungous dermatitis is necessary   however, neither parasites nor significant fungous infection can be demonstrated in greasy pig disease

TREATMENT

In the absence of a known causative agent there is no specific treatment available for greasy pig disease   Quite a variety of treatments have given apparently good results in some instances, however, no controlled experimental results are available to indicate the actual value   Large doses of penicillin have given satisfactory results in some cases   Recoveries have been credited to the use of various sulfa compounds   Manual methods of keeping the skin clean and free of excretions appear to be of value   Supportive nutritional treatment is often used in conjunction with therapeutic measures   Many pigs apparently live through a mild course of the disease without treatment and gradually recover to the extent that they become as thrifty appearing as pigs which have never been affected

The immunological significance of the fact that this condition is seen only in young pigs is unknown

REFERENCE

Jones L. D   1946   Exudative epidermitis of pigs   Amer Jour Vet Res 17 179

groups of muscles are affected varies widely There may be merely tremor of the head or nearly all of the muscles may appear to be involved, resulting in a jitterbug effect Unless nursing is interfered with to such a degree that starvation results, the mortality is low Most cases recover in 2 to 4 weeks In a few animals tremors persist until marketing age is reached

PATHOLOGICAL CHANGES

When paralytic symptoms result from pathological changes in the nervous systems the lesions are usually microscopic The exceptions are cases of tumors, abscesses and the rare instances in which gross inflammatory or degenerative changes are present The microscopic changes in the central nervous system consist of cellular mostly lymphoid, accumulations In addition to the usual evidence of inflammation, there may be more or less degeneration, neuronophagy, and gliosis Inflammatory changes may occur in either the white or gray matter In some cases, such as in Teschen disease, changes in the spinal cord are predominantly in the gray matter Sometimes the only lesion found is demyelinization of tracts of white matter in the spinal cord In some kinds of poisoning, such as by nitrophenide, the principal lesion is atrophy of the large nerve cells in the brain and cord

The changes in peripheral nerves accountable for paralysis or locomotory disturbances are usually either inflammatory or degenerative A well defined neuritis, characterized by lymphoid cell infiltration, is occasionally found This kind of change is most likely to involve the ganglia and roots of the spinal nerves Edema or neuromalacia of nerve trunks has been seen a few times in swine Chromatolysis of the ganglionic cells, loss of myelin, and axis cylinder degeneration are recorded findings in peripheral nerves, particularly in pantothenic acid deficiency

Bone lesions responsible for lameness or paralysis like symptoms are usually grossly obvious Osteomalacia is characterized by abnormally fragile bones which often fracture spontaneously The common sites of fracture are the femur and the pelvic girdle Fracture of vertebrae occasionally occurs in osteomalacia, but most of the vertebrae fractures or displacements probably occur in spondylitis Testing the fragility of the ribs at necropsy is a good way to diagnose osteomalacia

In rickets the bones are rubbery and there is widening of the proliferating cartilage at the ends of the diaphyses There may also be considerable bending of the long bones In other bone dyscrasias particularly that resulting from manganese deficiency the leg bones may be shorter than normal and there is not the widening of proliferating cartilage that occurs in rickets However the ends of some of the bones may appear enlarged and there may be considerable perosis

Arthritis is mainly of two types One occurs in young pigs while the other is in older animals The so called navel ill in young pigs is characterized by pus in the joints and sometimes by multiple abscesses elsewhere in the body In the type of arthritis prevailing in older swine the first pathologic change is synovitis At first the synovial membrane is congested and the villi are somewhat enlarged Fibrin clots are found occasionally, but there is ordinarily little or no increase of joint fluid Pus is rarely found in this type of arthritis

In later stages the joint capsule is thickened by fibrosis and the synovial villi show marked hypertrophy Erosion of the articular surfaces and pannus formation are found frequently Exostoses form in the vicinity of the affected joints These, together with fibrosis, cause marked enlargements Fibrous and bony ankyloses are rather common The hip, shoulder tarsal and carpal joints are frequently affected Spondylitis, characterized by exostoses and more or less ankylosis is commonly found This is the type of arthritis associated with swine erysipelas infection

DIAGNOSIS

The differential diagnosis of lameness and paralysis is frequently difficult Often

lesions Paronychia or panaritium some
times occurs in the absence of other recog
nizable symptoms or lesions of the known
specific diseases which affect the feet The
cause or causes of these foot lesions have
not been fully determined

CLINICAL SIGNS

The symptoms due to affection of the
nervous system are chiefly paralysis and
hyposensitivity However, hyperesthesia
may occur There may be general paralysis,
paraplegia or monoplegia Monoplegia
has been noted following the accidental in
jection of pseudorabies virus Hemiplegia
would be difficult to distinguish in swine
if it should occur Cases of varying degrees
of paralysis of the forelegs have been seen
apparently due to ganglionitis and neuri
tis involving the spinal nerves supplying
the front limbs

Posterior paralysis, such as occurs es
pecially in young swine, is characterized
by flaccid paralysis of the hind parts The
paralytic symptoms resulting from de
generation or inflammatory changes in the
spinal cord may be clinically indistinguish
able from those due to vertebral or other
bone fractures Occasionally a fracture in
the pelvic girdle, particularly in the ilium,
disables the hind parts of a young hog In
large hogs the fracture of a leg bone, a
vertebra, or the pelvic girdle may com
pletely disable the posterior parts of the
animal Usually it is impossible to detect
crepitation in the broken bones, particu
larly when they are high up in the leg, in
the pelvic girdle, or in the vertebral col
umn

The paralytic symptoms which result
from spondylitis accompanied by vertebral
fracture or dislocation, such as may occur
in brucellosis, usually develop gradually
In contrast, the symptoms resulting from
bone fracture due to abnormal fragility
come on suddenly Paralytic symptoms due
to brain lesions may be accompanied by
lethargy, nystagmus, torticollis, etc.

The lameness due to arthritis is variable
in its manifestations In young pigs there
often occurs rather rapid swelling of the
joints These swollen joints in young pigs

are usually soft, but may become firm after
many days The affected joint may be so
sensitive that the leg is disabled, even be
fore much swelling occurs In older
animals the arthritic joints usually do not
swell noticeably for several days, but finally
become more or less enlarged, very firm
and stiff In some cases the first sign of
arthritis is extreme sensitivity in the af
fected leg The animal may carry the leg
as if it were broken The lameness often
shifts from leg to leg The carpal and
tarsal joints are most conspicuously en
larged The carpi are usually symmetri
cally enlarged while most of the tarsal
enlargement is medial The condition
usually persists for weeks or months and
the animal becomes helpless A few indi
viduals recover

The peculiar condition known as goose
stepping' is characterized by extreme ex
tensor action in the hind limbs When the
animal walks, the hind legs are raised ab
normally high as they are moved forward
Some of these cases advance to the point
where there is considerable disability of
the hind parts In most instances the con
dition remains stationary or shows some
improvement More or less muscle atrophy
nearly always occurs, particularly in the
hind parts, whenever paralysis or other dis
ability conditions continue for some time

Myoclonia congenita or congenital trem
ors sometimes causes considerable loco
motory difficulty in newborn pigs It is
characterized by clonic spasms of the skele
tal muscles The extent to which muscles or

FIG 41 1 — Posterior paralysis of a 70 lb pig
which showed microscopic degeneration of the
posterior portion of the spinal cord but no evi
dence of vertebral fracture or malformation
Animal had been on alfalfa pasture (Photo
graph by H W Dunne)

of vitamin A or pantothenic acid is most likely to occur It is in connection with these deficiencies that prevention is of particular importance, since the resulting changes in the nervous system may be largely irreversible For a discussion of deficiencies and the sources of essential nutrients, see Chapter 49

REFERENCES

Albert, L , and Mendlowski, B   1956 Occurrence of a serosal disease in Wisconsin swine Vet Sci News (spring)

Bierer, B W   1956 Pasteurellosis Another baby pig disease Vet Med 51 91

Bohnstedt, G   1926 Mineral and vitamin requirements of pigs Ohio Agr Exp Sta Bull 395

Doyle, L P   1937 Posterior paralysis in swine Jour Amer Vet Med Assn 90 6 6

Follis R H   1948 The Pathology of Nutritional Disease Pub by Charles C Thomas Springfield, Ill

Glasser, K , Schmid, F , and Hupka, E   1944 Die Krankheiten des Schweines Verlag M and H Schaper, Hanover, p 82

Gualandi, G L   1955 Contributo allo studio eziologico della polsierosite fibrinosa dei suinetti Vet Ital 6 891 Abstract Vet Bull 26 733

Hjärre, A , and Wramby, G   1942 Om fibrinos serosaledinflamation hos svin Skand Vet Tidskr 32 257

Hughes, D L   1955 Arthritis in pigs The experimental disease induced by *Erysipelothrix rhusiopathiae* Brit Vet Jour 111 183

Hughes, J S , Aubel, C E , and Lienhardt, H T   1928 The importance of vitamin A and vitamin C in the ration of swine Kans Agr Exp Sta Tech Bull 23

Knott, S G   1956 Glasser s disease in pigs Queensland Agr Jour 82 33

Marsh, H   1931 The bacillus of swine erysipelas associated with arthritis in lambs Jour Amer Vet Med Assn 78 57

Miller, R D , Keith, T B , McCarty, M A , and Thorp, W T S   1940 Manganese as a possible factor influencing the occurrence of lameness in pigs Proc Soc Exper Biol and Med 45 50

Neher, G M , Doyle, L P , Thrasher, D M , and Plumlee, M P   1956 Radiographic and histopathologic findings in the bones of swine deficient in manganese Amer Jour Vet Res 17 121

Sikes, D , Neher, G M , and Doyle, L P   1955 Studies on arthritis in swine Amer Jour Vet Res 16 349

Usdin, M , Ferguson, L C and Birkeland, J M   1952 Experimental arthritis in swine following multiple injections with *Erysipelas rhusiopathiae* Amer Jour Vet Res 13 189

Ward, A R   1922 The etiology of polyarthritis in swine Jour Amer Vet Med Assn 61 153

the problem is to distinguish between nutritional deficiency and infectious disease A detailed history of how the animals have been fed and managed and the rate at which they have grown always helps in determining the nature of the trouble If the ration fed does not supply all the factors essential for developing and maintaining normal skeletal and nervous systems lameness or paralysis may result If the animals are deprived of sunlight and are so fed and managed that they do not get essential trace elements locomotory disability may result Rapid growth sometimes accentuates the effects of deficiencies

The skeletal defects such as occur in rickets and osteomalacia can usually be recognized by the findings mentioned under pathological changes Lesions in the nervous system are usually recognizable only by microscopic examination The specific diagnosis of the various infectious diseases which may cause paralysis or lameness requires animal inoculation or cultural examination suitable for the diagnosis of each disease The principal specific diseases to be considered in this connection are rabies pseudorabies Teschen disease, listeriosis, hog cholera, and swine erysipelas Nonspecific infections of the nervous system sometimes cause paralysis The comparatively rare cases of paralysis due to chemical poisons can usually be determined by finding out what the animals have eaten or with what they have been treated and by toxicological examination

TREATMENT

Treatments for the infectious diseases which cause paralysis are given under the discussions of these diseases Arthritis due to erysipelas appears to be difficult to prevent or cure Vaccination which may reduce death losses from acute swine erysipelas is not very effective in preventing arthritis Treatment should be applied early for best results Antibiotics, particularly penicillin have some curative value when used early After the disease has been present for some

time, treatment is not very effective No treatment or prevention is known for Glasser s disease

Of those nutritional deficiencies which are responsible for locomotory symptoms, the lack of calcium and phosphorus is perhaps the most common The feeding of adequate amounts of these elements is important particularly to growing swine and pregnant sows The calcium phosphorus ratio is considered to be of some importance However, making certain that enough calcium and phosphorus are fed is probably more important than the ratio between them The recommended ratio between calcium and phosphorus is 1 5 to 1 0 If the ration contains about 10 per cent of tankage or fish meal usually no additional calcium and phosphorus are needed

Whenever evidence of osteomalacia appears, treatment should be begun promptly so as to minimize skeletal damage The addition of bone meal to the ration at the rate of 1 to 2 per cent is an effective way to correct calcium and phosphorus deficiency Other sources of both calcium and phosphorus are defluorinated rock phosphate and dicalcium phosphate Calcium carbonate is a cheap source of calcium but it does not contain any phosphorus If symptoms of bone dyscrasis occur in animals kept indoors vitamin D should be supplied Fish oil is usually the most practical source of this vitamin

Of the trace mineral deficiencies which affect skeletal development, the lack of manganese is perhaps most likely to occur It is doubtful if this deficiency is of much general importance under farm conditions but it has been produced experimentally The addition of ¼–½ lb of manganese sulphate per ton of feed should supply the deficiency It may be well to supply choline too, in view of the part it has been found to play in preventing perosis in poultry

Of the other nutritional deficiencies which may cause paralytic symptoms a lack

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Heat Stroke, Sunburn, and Photosensitization

Heat Stroke (Sunstroke, Thermic Heat, Heat Exhaustion)

Heat stroke is a disorder of the heat regulatory mechanism which is a result of environmental hyperthermia

Dorland (1942) defines heat stroke as 'a condition caused by exposure to excessive heat, natural or artificial ' He divides heat stroke into three forms " (1) Thermic fever or sunstroke, (2) Heat exhaustion, and (3) Heat cramps ' Since the last form, heat cramps, is rarely if ever seen in swine, it will not be discussed in this chapter

Thermic fever, or sunstroke, is an acute condition characterized by sudden onset high temperature, and high mortality

Heat exhaustion is a mild form of heat stroke and is characterized by a gradual onset, depression, normal temperature, and a low mortality

To understand the causes of heat stroke, it is necessary to know, in a general way, how heat is produced and dissipated in the animal's body (Wiggers, 1919) Body temperature is increased by muscular activity, metabolism of foods, disease, drugs and adverse environmental conditions Heat is lost from the body by warming ingested food and inspired air, conduction convection, radiation, and water vaporization from the body surface and lungs. It is obvious that any acceleration of the factors that increase body heat production and any interference with the dissipation of body heat may result in hyperthermia

Our types and breeds of swine along with our system of rearing and marketing are predisposing causes of heat stroke

The lard type hog that has been popular in the United States and is still present in large numbers is ill adapted to withstand high temperatures His lung capacity is small on a per pound basis, when compared to some of our other domestic animals

In addition, he has a thick layer of fat which interferes with heat loss from the body surface He is often fattened under confinement and his respiratory and circulatory systems are not conditioned to withstand stresses such as he may meet before he is slaughtered

Another important factor is that the brachycephalic breeds have short noses and dished faces which tend to compress air passages This does not allow for the free passage of an increased volume of air that is required during periods of stress Old age and disease are other predisposing factors

Some of the immediate exciting causes of heat stroke are driving or handling hogs in very hot weather, crowding them in tight quarters with inadequate ventilation, far rowing during hot weather, providing inadequate water at times and allowing a few hogs to the herd during very hot weather

Sunburn

Sunburn is a dermatitis caused by the action of ultraviolet rays upon the unprotected skin. It is a problem in the breeds of swine which have white skins. Other breeds may be affected but to a much lesser degree. Sunburn occurs when very young pigs with tender skins are exposed to bright sunshine and when white swine of any age are placed in bright sunshine without a period of acclimation. Swine do not become sunburned indoors because window glass does not permit passage of the ultraviolet rays.

CLINICAL SIGNS

The severity of signs is determined by the brightness of the sun, length of exposure, and sensitivity of the skin. The signs usually appear within several hours after exposure. An erythema develops due to vasodilatation of peripheral capillaries. In addition there is an increase in capillary permeability with resulting edema. Affected swine are warm to the touch and show evidence of pain. They walk very carefully and squeal when they contact any object. After several days thin layers of skin peel off and the hogs are relatively immune to the effects of ultraviolet rays. Much of the immunity is probably due to

a thickening of the outer layers rather than tanning.

Very young pigs may be burned so severely that their ears and tails will necrose and slough.

Sunburn is rarely fatal in swine.

DIAGNOSIS

Diagnosis is based on a history of unacclimated swine being exposed to bright sunlight, an erythema and edema of the skin.

The absence of a photodynamic agent and the relatively mild nature of the condition serves to differentiate it from photosensitization.

TREATMENT

Providing protection from the sun for a few days is usually all that is required when swine become sunburned. In severe cases bland oils may be applied to the skin.

PREVENTION

Protection from bright sunlight should be provided for baby pigs. Animals with white skin should be exposed to bright sunshine for short periods of time until their skins acquire some protection. All hogs should have access to shade during hot weather.

Photosensitization

The term photosensitization indicates an increased or unusual sensitivity to light. In swine, the condition is characterized by a superficial necrosis of the unpigmented or lightly pigmented areas of skin. In general the appearance of the symptoms of photosensitization is dependent upon two factors: (1) a hog must ingest a specific sensitizing substance known as a photodynamic agent, and (2) the animal's skin must be exposed to sunlight.

Bourne (1953) defines a photodynamic agent as any chemical substance whose presence in the skin or other tissues is cap-

able of absorbing certain wave lengths in sunlight and converting the radiant energy into molecular energy which is passed on to other molecules and in the presence of molecular oxygen sets up the tissue changes characterizing the clinical aspects of the disease.

We have numerous known photodynamic agents in the world today, and no doubt more will be found in the future. Clare (1952) has stated that 55 plants have been proven or are at least suspected to contain photodynamic agents. Some of the more common plants are alfalfa, red clover,

and permitting undue sexual excitement due to inadequate isolation of animals in estrus

CLINICAL SIGNS

At the onset, affected animals are depressed and seek shade and water As the condition develops, they become dyspneic salivate profusely, exhibit open mouth breathing, and may become extremely restless The temperature may rise to 110° F or even higher They may approach a state of frenzy and become ataxic constantly changing positions

Oftentimes the animals will assume a sitting position As anoxia develops due to circulatory failure the visible mucous membranes will become cyanotic Eventually the animal becomes comatose, and death occurs within a few hours in severe cases if satisfactory treatment is not administered Occasionally animals recover from the acute signs of the disease but are unable to tolerate hot weather They may also have permanent mental derangement

PATHOLOGICAL CHANGES

The carcass is usually well covered with fat. A blood stained foamy discharge is often exuding from the nostrils Much of the viscera is edematous and hemorrhagic This is particularly true of the lungs The central nervous system is edematous and there may be destruction of nerve cells in the cerebral cortex

DIAGNOSIS

Diagnosis is often difficult because heat stroke may accompany many diseases It is imperative to secure a complete history and conduct a thorough physical examination The presence of any of the predisposing factors and exciting causes in very hot weather along with the described symptoms justifies a diagnosis of heat stroke

TREATMENT

Prompt reduction of body temperature is indicated when heat stroke is diagnosed but caution must be observed in spraying or pouring very cold water on the back of the hog suffering from heat stroke

Abundant amounts of cool water should be used on the floor surrounding the affected animal His legs, underline, and head should be bathed with cool water

In severe cases ice packs may be used on his head and legs Fans should be used to facilitate proper circulation of air In addition to lowering the body temperature, it is indicated to administer stimulants or depressants, dependent upon the condition of the animal If he is in a state of frenzy, mild sedation should be cautiously administered If the animal is comatose and circulatory failure is eminent, stimulants and oxygen should be given

Following a decline in temperature and improvement in the animal's general condition, it is advisable to observe him very closely for the next 24 hours since the symptoms of heat stroke may reappear after treatment is discontinued

PREVENTION

Prevention of heat stroke consists of maintaining hogs in as comfortable an environment as practical without subjecting them to any undue stresses

Some of the more important preventive measures to observe during hot weather are

1 Provide adequate shade, water, and ventilation
2 Provide a well balanced laxative diet
3 Move or handle hogs during the early morning or evening hours
4 Dispose of breeding animals with chronic respiratory diseases
5 Provide separate quarters for males and females during the breeding season to prevent undue sexual excitement
6 Maintain sows in medium condition rather than very fat at farrowing
7 Acclimate hogs to very hot sun by exposing them for short periods or by giving them the initial exposure during more moderate days
8 Make additions to the herd during cool periods of the day, and, if fighting occurs, remove the offenders

lamp black retards penetration of epidermal layers by the sun's rays

PREVENTION

When swine are pastured, it is not always possible to prevent photosensitization Close observation of the animals and removal from the pasture at the first signs of photosensitization will prevent serious attacks If it is necessary to pasture hogs on forage containing photodynamic agents, it is advisable to confine them indoors during the day and allow them to graze at night.

Plants which have grown very tall and have been trampled down and damaged should not be fed to swine if photosensitization is a problem

Phenothiazine should not be given to swine under 70 days of age (Swales 1912)

REFERENCES

Heat Stroke

DORLAND, W A N 1942 The American Illustrated Medical Dictionary 19th ed W B Saunders and Company, Philadelphia and London p 1391

WIGGERS, C J 1949 Physiology in Health and Disease 5th ed Lea and Febiger Philadelphia p 974

Photosensitization

BOURNE R F 1953 Photosensitization No Amer Vet 31 173

CLARE, N T 1952 Ruakura Anim Res Sta Dept Agr No 3 Hamilton New Zealand

McCLYMONT G L 1955 Possibility of photosensitization due to ingestion of aphids Australian Vet Jour 31 112

KLUSSENDORF, R C 1954 Photosensitization No Amer Vet 35 665

SWALES W E ALBRIGHT, W D FRASER L AND MUIR G W 1912 Photosensitization produced in pigs by phenothiazine Canad Jour Comp Med and Vet Sci 6 169

ladino clover, alsike clover, rape, vetch, buckwheat, oats, and St. John's wort. The nothiazine is a photodynamic agent. McClymont (1955) has reported that some aphids contain a fluorescent pigment which may promote photodynamic hemolysis.

The pigment phylloerythrin, which is one of the end products of chlorophyll catabolism, is the most common photodynamic agent. Usually phylloerythrin is excreted by the liver, but, if an excessive quantity is present or the liver is not functioning properly, large amounts of this fluorescent pigment may appear in the tissues. Partial decomposition of plants before ingestion apparently increases the amount of phylloerythrin which is produced.

CLINICAL SIGNS

Four factors which influence the onset of symptoms are: amount of photodynamic agent in the skin, length of exposure to bright sunlight, color of skin, and the color and amount of hair covering the skin. Usually the symptoms appear within the first week after swine begin grazing on forage containing photodynamic agents.

The first signs are erythema, edema, pain, and slight elevation of temperature. If the pigs are spotted or have white points, there will usually be sharp demarcation between the pigmented and non pigmented areas with only the unpigmented areas being affected. Early signs are followed by exudation of serum which dries and mats down the hair. The ears are often thickened, the conjunctiva congested, and the eyelids matted together. Pain is evidenced by the animal's careful gait and suddenly dropping to the ground with rear limbs extended posteriorly. Hemoglobinuria may be present. After a few days the skin becomes very dry, hard, and fissured. At this time, pigs show extreme pruritus, rubbing on the fence, buildings, or any solid object they can contact.

In approximately one week the superficial layers of necrotic skin begin to separate from the underlying tissues. The edges begin to separate first at the fissures and the strips begin to curl. These strips

eventually drop off or are rubbed off, exposing a partially healed area devoid of hair. In most cases these areas again become covered with hair but due to the scarring of the skin the haircoat is rough and staring.

The mortality is low but economic losses occur because swine lose weight during the acute phase of the condition and they often do not gain satisfactorily for some time after the decline of symptoms.

GROSS LESIONS

The most marked lesion observed at necropsy is a necrosis of the superficial layers of unpigmented or lightly pigmented skin. In severe cases the ears and tail may be necrotic and in the process of sloughing. It is also possible, but rare, for the liver and kidneys to be enlarged and necrotic.

DIAGNOSIS

The diagnosis is made on the basis of superficial necrosis of unpigmented or lightly pigmented areas of skin, ingestion of a photodynamic agent, and exposure to sunlight.

Photosensitization is most likely to be confused with sunburn and erysipelas. The ingestion of a photodynamic agent and the severity of photosensitization are of major value in differentiating it from sunburn. Erysipelas is marked by high temperatures and skin necrosis which is non selective, relative to pigmentation. In addition the herd history and swollen joints are of value.

TREATMENT

In mild cases, treatment consists of placing the animals in darkened quarters and removing from their diet the ingredient which contains the photodynamic agent. In more severe cases, it may be necessary to give them a laxative and apply some protectant to the skin. Non drying oils aid in softening the crusts, thus decreasing irritation to the skin and aiding in the removal of the necrosed tissue.

Klussendorf (1951) states that kaolin bismuth mixtures prevent necrosis and that

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The leg malformations were fusion
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They believed the factor or factors
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good quality

S SYSTEM MALFORMATIONS

ral nervous system defect char
in part as *external hydrocephalus*
reported in Duroc Jerseys and
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have enlarged heads, light coat
hort tails The bristles are light
silvery sheen The tail is short
There is always an accumu
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accumulation of fluid is great
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Catlin Mark is present
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ALFORMATIONS

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In these cases there is partial or complete
closure or absence of a portion of the ali
mentary tube The animals are usually
born alive but die within a few days The
author observed a pig which had lived 3
weeks with atresia ani The pig suckled
but eventually became greatly bloated and
depressed Knilans (1913) encountered a
litter of 6 pigs, 3 of each sex, in which the
females had no anus but due to failure
of partitioning between the rectum and
vagina defecation was accomplished
through the vulva It is believed by ge
neticists that these defects are probably the
result of complex inheritance

CIRCULATORY SYSTEM MALFORMATIONS

Reports of disturbances in the develop
ment of the circulatory system of pigs are
extremely rare There is an occasional re
port on unusual arrangements of the great
vessels but these abnormalities are usually
discovered by instructors of laboratory
classes in embryology and are not signifi
cant enough to warrant description here

Of more importance perhaps is a defect
in the coagulation mechanism of swine
blood which occurred in Missouri (Muhrer
et al, 1912) In a herd of swine severe
bleeding occurred whenever certain
animals were injured as when their ears
were notched rings were put in their
snouts males were castrated teeth became
loose, joints were bruised, or when the
vagina was traumatized in coitus or at
parturition It was a transmissible defect
but not sex linked as is human hemophilia
In swine it is transmissible to both sexes

The presence of this defect in the blood
coagulation mechanism can be detected by
determining the fibrin precipitation time
in diluted plasma This is considered to
be a reliable test for the abnormality The
injection of a globulin fraction prepared
from normal blood reduces the coagulation
time of the blood of abn rmal animals

GENITAL MALFORMATIONS

In a study of the generative organs of
2000 sows and gilts by Wiggins and his
colleagues (1940), aplasia and hyps at

RUSSELL A. RUNNELLS, D V M , M S

The Upjohn Company
Michigan State University, emeritus

Malformations

In the course of the embryonic develop
ment of pigs it is reasonable to suppose
that deviations from normal may occur as
in other animals and result in the same
kinds of malformations However, the lit
erature is not burdened with reports on
such malformations Just as in other
animals a malformation may involve an
organ or a part of the body (anomaly) or
be much more extensive, so that great de
formity of the individual occurs (monster
or monstrosity)

Obviously the degree of malformation
may be variable It may be so slight that
the value of the animal for food is not
affected yet great enough to disqualify the
animal as a breeder On the other hand,
the disturbance in development may be
great enough to interfere with the growth
or the health of the pig or even extensive
enough to cause its death prenatally or
within a few days or weeks postnatally

CAUSES OF MALFORMATIONS

The causes of malformation are not al
ways known Some have their origin in
the genes of the chromosomes and are
therefore heritable By far most of those
reported in swine have been heritable A
few have been attributed to nutritional de
ficiencies, infections including vaccination
with modified live hog cholera virus, and
trauma Most of the malformations begin
to make their appearance in the embryo
within the first few days or weeks after fer

tilization of the ovum Only a few prob
ably have their origin in the later stages
of pregnancy

HERITABLE MALFORMATIONS

Heritable defects are the result of defi
nite elements in the germinal makeup
They are transmitted in accordance with
the Mendelian laws of inheritance and in
many instances their mode of inheritance
has been definitely determined As in the
case of other heritable characters certain
defects are inherited in a simple fashion
while others are due to larger gene com
plexes Most hereditary defects appear to
be simple, recessive, autosomal characters
This means that the genes responsible for
them are not in the sex determining chro
mosomes and that heterozygous animals
that is those carrying a single mutant gene
do not show the malformation however
when two such carriers are mated and pro
duce offspring, about 25 per cent of the
progeny have the hereditary malformation
About three of the offspring will be normal
and one will present the disturbance in de
velopment This, of course, is the familiar
Mendelian ratio In swine these recessive
mutants result when a carrier boar is
mated with his own gilts or with gilts of
some other carrier Since in either case only
about one half of these gilts will carry
the recessive gene, the other half can pro
duce only normal pigs. The resultant ratio
of normal animals to malformed animals

[567]

muscles by feeding pregnant sows a ration deficient in a factor or factors necessary to support normal reproduction and lactation The leg malformations were fusion of the digits and contraction of the tendons They believed the factor or factors missing in the ration are present in alfalfa meal of good quality

NERVOUS SYSTEM MALFORMATIONS

A central nervous system defect characterized in part as *external hydrocephalus* has been reported in Duroc Jerseys and Poland Chinas Pigs born with this malformation have enlarged heads, light coat color, and short tails The bristles are light red with a silvery sheen The tail is short or absent There is always an accumulation of fluid in the arachnoid space of the brain even though the quantity is not great enough to cause enlargement of the head If the accumulation of fluid is great enough, there is distinct bulging of the frontoparietal region At this place a defect similar to ' Catlin Mark ' is present

If the pigs are born alive, they stand with difficulty Their joints are stiff, and if they are forced to stand, they squeal as though in pain, and quiver If forced to move, there is incoordination The jaws are closed so that nursing is impossible If the pigs are born alive, they die within a day or two A single recessive autosomal gene has been determined to be responsible for this defect.

Of lesser importance from the standpoint of swine raising are the anomalies which are occasionally described in the literature such as that of a pig embryo with bifid notochord, biaxiate brain, and paired hypophyses and congenital abnormality of the Purkinje cells, but this discussion should be directed more to the malformations of greater economic importance

DIGESTIVE SYSTEM MALFORMATIONS

Now and then in the literature there are reports of defects in the development of the gastrointestinal tract, particularly in the caudal half Among such malformations are *atresia* of the ileum and of the anus

In these cases there is partial or complete closure or absence of a portion of the alimentary tube The animals are usually born alive but die within a few days The author observed a pig which had lived 3 weeks with atresia ani The pig suckled but eventually became greatly bloated and depressed Knilans (1913) encountered a litter of 6 pigs, 3 of each sex, in which the females had no anus but due to failure of partitioning between the rectum and vagina defecation was accomplished through the vulva It is believed by geneticists that these defects are probably the result of complex inheritance

CIRCULATORY SYSTEM MALFORMATIONS

Reports of disturbances in the development of the circulatory system of pigs are extremely rare There is an occasional report on unusual arrangements of the great vessels but these abnormalities are usually discovered by instructors of laboratory classes in embryology and are not significant enough to warrant description here

Of more importance perhaps is a defect in the coagulation mechanism of swine blood which occurred in Missouri (Muhrer *et al*, 1942) In a herd of swine severe bleeding occurred whenever certain animals were injured as when their ears were notched, rings were put in their snouts, males were castrated, teeth became loose, joints were bruised, or when the vagina was traumatized in coitus or at parturition It was a transmissible defect but not sex linked as is human hemophilia In swine it is transmissible to both sexes

The presence of this defect in the blood coagulation mechanism can be detected by determining the fibrin precipitation time in diluted plasma This is considered to be a reliable test for the abnormality The injection of a globulin fraction prepared from normal blood reduces the coagulation time of the blood of abnormal animals.

GENITAL MALFORMATIONS

In a study of the generative organs of 5 088 sows and gilts by Wiggins and his colleagues (1950), *aplasia* and *hypoplasia*

is then not 3 1 but 7 1 Naturally the reason malformations in swine are so seldom seen is that swine breeders usually avoid further use of a boar that is a proven carrier of an anomaly and discard normal full siblings of affected animals

SKELETAL MALFORMATIONS

A heritable lethal factor causing *legless pigs* was reported by Johnson (1940) In the herd in which this malformation appeared, 2 boars, both heterozygous for the causative gene sired some pigs in which the bones of the shoulder and pelvic girdles were normal or nearly so but all the leg bones were missing The ratio of normal pigs to legless pigs was 207 25 This conforms closely to the 7 1 expected ratio which would have been 203 29 These pigs were born alive and at the outset were as vigorous as the normals but died within 3 days

A heritable malformation which not always but usually is fatal is a defective development of the skull In most cases of fected pigs die within an hour The defect occurs in the frontal region and usually consists of an arrest in development of the frontal and parietal bones on the midline As a result, a hole is left in the skull at about the place of intersection of 2 lines each drawn from an ear to the opposite eye The defect has been called *"Catlin Mark"* The skin over the opening may be covered with hair At the Calif Agricultural Experiment Station (Hughes and Hart, 1934), where this has occurred, the

character was a single recessive from both parents and occurred in Poland Chinas

Cleft palate, a recessive character, has been reported in swine As in other animals it is due to failure of the lateral palatine processes to develop completely These two shelflike processes, outgrowths from the medial aspects of the maxilla fail to meet and fuse on the midline of the oral cavity It is transmitted as a simple recessive character Since the defect prevents nursing, the pigs soon die

In the swine raising sections of the United States the introduction of modified (attenuated live) hog cholera virus for immunization against hog cholera was accompanied by a peculiar syndrome in offspring of some vaccinated pregnant sows The syndrome was characterized by ascites, anasarca, edema of the mesocolon and perirenal tissues, hydropericardium and hydrothorax, mottling of the liver, and the following malformations asymmetry of the head narrowing of the head, lengthening and twisting of the snout, and malformation of the limbs (Fig 43 1) Experimentally the condition was produced in Minnesota by injecting modified live hog cholera virus into sows on the 14th to 16th day after mating (Sauter et al , 1953) The Minnesota researchers believe that the presence of live virus in the body early in pregnancy may interfere with the development of some organs

In Wisconsin, Ross et al (1944) produced malformations of the limbs and paralysis with constant tremor of the
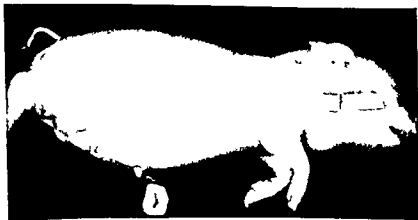
FIG 43 1 — Fetal distortion as the result of subcutaneous edema and ascites Dam had been injected with modified live virus 16 days after breeding Fetus was 105 days old at time photograph was taken and was the only pig alive in the litter (Photograph courtesy J H Sauter, G A Young, A J Luedke, and R J Kitchell, University of Minnesota and Hormel Institute)

genic tissue The ovary had the structure of that of a normal sow about 10 days post ovulation The uterus was in the early phase of luteal stimulation

In a case of true lateral hermaphroditism reported by Brown (1944) an imperfect ovary was attached to the broad ligament on the left side The oviduct and left horn appeared normal The right horn was small and had no tube The two horns fused into a body but the cervix and vagina were imperfect and terminated in fat in the pelvic cavity On the left side the male organs were represented by an imperfect testis which was located just outside the inguinal canal It had a small epididymis and an undeveloped vas deferens which ended in abdominal fat Externally there were normal labia but no connection with the vagina The clitoris was large with the urethral opening on its dorsal surface The prepuce was normal in shape but had no opening The teats were small like those of the male and no mammary gland tissue was recognizable

Brambell (1929) described the histology only of the gonads of a pig which may have been a *unilateral hermaphrodite* Such an animal has an ovary and a testis on one side and either an ovary or a testis on the other Brambell s case had a testis on the left side and an ovotestis on the right

Fourie (1935) in South Africa described a false *masculine hermaphrodite* pig which had two testes each with an epididymis and a vas deferens The vas deferens whose lumen was small, led to a point in the vicinity of the external urethral orifice and terminated The horns of the uterus which were poorly developed were attached to the distal end of the corresponding epididymus There were no oviducts or ovaries The cervix was of the usual length but its wall was thin and its canal ballooned The vagina was poorly developed The vulva was normal with a large clitoris The testes were nonfunctional and the Leydig cells hypoplastic This type of hermaphroditism is also called the *transverse* and is characterized by external organs indicating one

sex and by the presence of gonads of the opposite sex

HERNIA

The common hernias in swine are in guinal scrotal, and umbilical These are so common and of such economic importance that they have been a problem for extensive investigation at the Wisconsin Agricultural Experiment Station (Warwick 1926)

In the study in Wisconsin up until weaning time 1 68 per cent of the male pigs had inguinal hernia and 0 6 per cent had umbilical Among females 1 16 per cent had umbilical hernia In the males it was believed that the anatomical defect which resulted in the hernia was a large internal vaginal ring and a low degree of tensity of the tunica vaginalis

The testis in the fetal pig normally reaches the lower aspect of the inguinal canal between 80 to 90 days after fertilization of the ovum At 100 days or later the testes descend into the scrotum During the next 12 to 18 days before birth the testes complete their development By this time the tunica vaginalis must be well enough developed to withstand pressure during and after birth After the testis descends into the scrotum the canal closes and the intestines are held back in the abdominal cavity If the inguinal ring is unduly large hernia of the intestine may occur It may be detected immediately after birth or may not occur until sometime within the first month If the condition is unilateral it occurs more frequently on the left side

In the Wisconsin experiment herniated boars were mated to closely related females These breeders came from a herd in which 7 49 per cent of the male pigs had hernia in the year the project was begun Prior to this the parent herd had a 3 year record of 1 19 per cent herniated male pigs After mating the herniated males with closely related females the hernia rate jumped to 11 28 per cent in the males in the first generation in the second generation to 12 per cent, and in the third generation to 13 18 per cent

of parts were observed in only 0 7 per cent of the cases, and duplication of parts in only 0 1 per cent The most common aplasias and hyperplasias involved the horns of the uterus The defect was usually unilateral and in these cases did not interfere with breeding efficiency, although it would seem reasonable that the size of the litter might be reduced In one case there was no cervix, in another an absence of horns, cervix, and vagina, and in two the vulvae were missing Only three of the females had duplication of parts, in two there were two cervices, and in another a portion of a horn was bifid These duplications did not interefere with pregnancy

In the same study 3,476 sows and gilts were examined for *persistence of male ducts* in the broad ligament along the uterus Such persistence occurred in 8 9 per cent of the animals In some cases the duct ran along in an unbroken line where as in others it resembled a string of beads This form of persistence of fetal structures occurred unilaterally and bilaterally, with combinations of continuous and segmented tubes The persistence of this fetal male structure did not seem to interfere with the breeding efficiency of the females

Cryptorchidism in swine has been studied quite thoroughly at the Missouri Agricultural Experiment Station (McKenzie, 1931) Attention to the problem there was stimulated by the occurrence of the condition in 10 of the herd boars Five had double cryptorchidism and 5 single In unilateral cases the left testis is usually involved One boar failed to develop male characteristics

To determine whether the condition was heritable in the Missouri herd, a normal male was selected from a litter in which other males had the condition This boar was mated to 12 sows The boar was unrelated to the sows but 4 of the sows were related to each other, one was the dam of 3 of the others The 12 sows farrowed 107 pigs of both sexes In the litters of 5 sows, 10 cryptorchids appeared Four of these 5 sows were the related ones mentioned above After various combinations of mat

ings among these animals it was concluded that cryptorchidism is probably inherited in much the same fashion as is herniation

The writer has been able to find in the literature only one report of a case of *complex hermaphroditism* in swine Such an animal has the external and internal genital organs of both sexes

In the literature available to the author there are reports of about thirteen cases of *true lateral hermaphroditism (gynandromorphism)* in swine In this form of intersexuality a male gonad is present on one side and a female on the other In the embryo the male hormone stimulates the development of the Wolffian duct from which several of the male sex organs develop and the female hormone stimulates the Mullerian duct from which several of the female sex organs arise These hormones are produced in the interstitial cells of the gonads In cases of intersexuality it is believed that the hormone which should dominate in the development of the particular sex organs is deficient In the absence of its governing influence the hormone in the rudimentary gonad of the opposite sex exerts its influence so that structures of both sexes become prominent

In true lateral hermaphroditism, as stated previously, a male gonad is present on one side and a female gonad on the other Outside of this there is no set anatomical pattern The other sex organs may in part be well developed, poorly developed, or lacking In a case reported by Nielson (1941) there was a testis on the right side and an ovary on the left The left horn of a somewhat enlarged uterus had a normal oviduct but the right horn had an abbreviated oviduct which terminated blindly near the testis There was a normal cervix On the right side the male organs were represented by the testis, an epididymis, and a vas deferens Unfortunately Nielson did not see the external genitals of this case Histologically he found the testis to be of the cryptorchid type There was hyperplasia of the interstitial tissue and an absence of spermato

nancy results in a progressive reduction in offspring affected by the malformation From the standpoint of the embryo it appears that the critical time for vitamin A deficiency to occur in a pregnant dam is at the period when organ formation is active tively taking place Embryologists believe that the formation of the secondary organ zer within the embryo has been interfered with In the case of microphthalmia the in terference apparently occurs at the time of evagination of the optic vesicle Evagina tion is incomplete and tissue differentiation in the eye is only partial

In Denmark Sweden, England and the United States cases of microphthalmia in pigs have been associated with vitamin A deficiency in sows It has been most preva lent in pigs in drought seasons

Microphthalmia has been experimentally produced by Hale (1935) In his experi ment 42 pigs in 4 different litters were born blind Some of them also had other malformations such as cleft palate cleft lip, accessory ears and arrested ascension of the kidneys He ruled out genetic fac tors as a cause The condition appeared in the offspring of sows that were depleted of vitamin A before breeding and were fed a vitamin A deficient ration for 30 days after breeding This 30 days constitutes the period during which the eye develops

Maneely (1951), however, was not en tirely convinced that the malformation was the result of vitamin A deficiency because he observed it among 27 pigs which were farrowed by 1 sows that were bred to the same boar Seven of the pigs had the mal formation

MONSTERS

Most animals that have disturbances in development extensive enough to be classed as monsters are ones in which there is in complete twinning One exception to this is the monster called *acardiacus* It is a twin arising from one fertilized ovum completely separate from its mate but much smaller Since it derives its blood supply from the placenta of the more or less nor mal twin it is termed a parasite It has no

heart or only a vestige of one It may be devoid of a head and may have a very rudimentary thorax but a well formed pel vis Such a monster is designated *acardiacus acephalus* If the mass which represents the fetus is only a ball of rudimentary organs having no heart and covered with hair the designation is *acardiacus amorphus* The author has seen such monsters in cattle but never in swine Furthermore he can find no reference to any in the available litera ture

The more commonly observed monsters are those which are examples of incom plete twinning They are derived from a single fertilized ovum which in the cleavage or early gastrula stage of its development was disturbed in some way so that the de velopmental pattern became that of two individuals joined The factor or factors responsible for the incomplete twinning are not known

Reports of incomplete twinning in swine are exceedingly scarce in veterinary litera ture even though specimens of such condi tions are commonly found in the anatomy and pathology laboratories of most veteri nary colleges These double monsters in their uterine development are enclosed in a single chorion If the twinning involves the posterior part of the body both parts have the same sex because they are con joined identical twins The conjoined fetuses are generally united ventrally that is, belly to belly The part of the body which undergoes the twinning may be the anterior with the posterior still a single individual (pygopagus), or the twinning may involve the posterior part with the anterior still a single individual (cranio pagus or cephalothoracopagus depending upon the extent to which the fetus is a single individual) To complete the desig nation of the monster it is necessary to indicate how much twinning occurs in the part of the body affected As an example the number of eyes ears mouths and four limbs must be taken into consideration when applying a name to a pig fetus that has anterior twinning

In the museums of veterinary colleges

On the basis of these results the Wisconsin researchers concluded that hernia in pigs is a heritable character and stated their conclusions as follows   Herniated boars result from the double recessive geno type (h h h h) while the females of the same construction are normal

WATTLES

An interesting but harmless defect in development was reported by Roberts and Morrill (1944) in Illinois The defect consists of a pair of cutaneous teatlike appendages which hang down on each side from the mandibular region They are called wattles but look something like the leader or caruncle on a gobbler The wattle has a central core of fibrocartilage which has a roughened knoblike proximal end embedded in the subcutaneous tissue of the under surface of the jaw The cartilaginous core is covered with dense connective tissue and skin

The circumstances under which this condition occurred are interesting A grade Hampshire boar that had wattles sired several litters from sows that did not have the defect The owner of the boar reported that one half of the offspring had wattles This suggested dominence of the character and a heterozygous condition of the boar

The authors state that wattles are inherited as a Mendelian dominant character in many breeds and strains over the earth They cite an instance in which there were 84 pigs in 9 litters all from normal sows Among the 65 pigs which lived 29 had wattles so it appears that the defect is inherited as a simple dominant character

MICROPHTHALMIA

A malformation which has attracted attention in England Denmark Sweden and the United States is microphthalmia a condition associated with blindness (Fig 43 2) In all cases the eyes are small and their state of development quite variable Parts of the eye may be missing or if present they may be very imperfectly formed For instance, a very rudimentary lens may be present or the lens may be completely lacking If only the primordium of the lens is present it may be imbedded in a mass of connective tissue Most of the remainder of the eye may be represented by a mass of connective tissue in which smooth muscle and vesicles of pigmented (melanin) cuboidal choroidal cells are laid down in a helter skelter fashion In other cases the sclera is poorly formed and is lined within with a papillated choroid which is imperfectly formed There may be islands or strips of retina around a central lumen or in a dense mass of connective tissue In these strips and islands of retina rods and cones may be present or only rods Portions of the vitreous body may be present

This form of eye defect in pigs is believed to be the result of the transmission of a lethal gene or the effect of vitamin A deficiency in the pregnant sow It has been shown in rats that pregnant females with vitamin A deficiency have offspring which exhibit various congenital defects among them eye lesions like these in pigs Administration of vitamin A to such females at progressively earlier times during preg

FIG 43 2 — Pig born without eyes   All of the litter of 10 pigs were born blind as the result of a maternal vitamin A deficiency (Photograph courtesy Fred Hale Texas A & M College Agricultural Experiment Station)

SHAKER, R. B 1956 The persisting right sixth aortic arch of mammals with a note of fetal coarctation Anat Rec. 125 171

STEVENSON, J R 1954 A case of atresia of the ileum with a divided kidney in the foetal pig Anat Rec 118 211

THURINGER, J M 1919 The anatomy of a dicephalic pig (monosomus diprosopus) Anat Rec 15 359

WARWICK, B L 1926 A study of hernia in swine Wis Agr Exp Sta Res. Bull 69

WARWICK, E J CHAPMAN, A B, AND ROSS B 1943 Some anomalies in pigs Jour Hered 31 319

WATT, J A, AND BARLOW, R M 1956 Microphthalmia in p glets with avitaminosis A as the prob able cause Vet Rec 68 780

WIGGINS, E. L, CASIDA, L E AND GRUMMER R. H 1950 The incidence of female genital abnor malities in swine Jour Anim Sci 9 3

WILLIAMS, S R, AND RAUCH, R W 1917 The anatomy of the double pig (syncephalus thora copagus) Anat Rec. 13 273

WILSON, J G, ROTH, C B AND WARKANY J 1953 Syndrome of A deficiency anomalies. Amer Jour Anat 92 189

YOOLICK, B L. 1955 True lateral hermaphroditism in a pig Tex Rep Biol and Med 13 187

there are generally pig fetuses also in which the twinning was almost complete They are united ventrally either at the thorax (thoracopagus) , at the thorax, the cervical region, and to some extent at the head (prosopothoracopagus) , or at the thoracic and lumbar regions (rachipagus)

The double monsters just described are those with symmetrical development Oc

casionally one may be large and the other small and embedded in the larger The larger is termed the autosite and the smaller the parasite By a stretch of the imagination one can conceive of a teratoma of the testis or ovary as belonging to this same category of monsters since it is generally believed that teratomas of the gonads are included twins

REFERENCES

BAKER, J R 1925 Assymetry in hermaphrodite pigs Jour Anat 60 374

BAUMGARTNER, W J 1928 A double monster pig — cephalothoracopagus monosymmetros Anat Rec 37 303

BENDIXEN, H C 1944 Littery occurrence of anophthalmia or microphthalmia together with other malformations in swine, presumably due to vitamin A deficiency of the maternal diet in the first stage of pregnancy and the preceding period Acta Path et Microbiol Scand 21 605

BLUNN, C T AND HUGHES, E H 1938 Hydrocephalus in swine, a new lethal defect Jour. Hered 29 203

BRAMSELL, F W R 1929 The histology of an hermaphrodite pig and its developmental significance Jour Anat 63 397

BROWN, C E 1944 Lateral hermaphroditism (gynandromorphism) in a pig Vet Med 39 456

CAREY, E 1917 The anatomy of a double pig, syncephalus thoracopagus, with special consideration of the genetic significance of the circulatory apparatus Anat Rec 12 177

CHIDESTER, F R 1914 Cyclopia in mammals Anat. Rec. 8 355

CORNER, G W 1921 A case of lateral hermaphroditism in a pig with functional ovary Jour Urol 5 481

CREW, F A E 1921 Hermaphroditism in the pig Jour Obst. and Gynaec Brit Emp 31 1

EATON, O N 1937 A summary of lethal characters in animals and man Jour Hered 28 320

FOURIE, J 1935 False masculine hermaphroditism in a pig Onderstepoort Jour Vet Sci 4 573

GEORGE, W C. 1914 A pig embryo with bifid notochord, biaxiate brain and paired hypophyses Anat Rec. 89 107

GREGG, R E. 1916 An arterial anomaly in the fetal pig Anat Rec 95 53

HALE, F 1935 The relation of vitamin A to anophthalmos in pigs Amer Jour Ophthal 18 1087

HUGHES, E. H, AND HART, H 1934 Defective skulls inherited in swine Jour Hered 25 111

HUGHES, W 1927 Sex intergrades in foetal pigs Biol Bull 52 121

HUTT, F B 1934 Inherited lethal characteristics in domestic animals Cornell Vet 21 1

——— 1916 Some hereditary abnormalities of domestic animals Cornell Vet 36 180

JOHNSON, L E. 1910 Streamlined pigs Jour Hered 31 239

JORDAN H E, DAVIS, S J, AND BLACKFORD, S D 1923 The operation of a factor of spatial relationship in mammalian development as illustrated by a case of quadruplex larynx and triplicate mandible in a duplicate monster (craniopagus). Anat Rec. 26 311

KNILANS A J 1913 Cloaca in three pigs. Jour Amer Vet. Med Assn 103 27

KOLODNER, J E. 1955 True hermaphroditism in a hog Jour Amer Vet Med Assn 126 223

MCKENZIE, F F 1931 Cryptorchidism in swine Mo Agr Exp Sta Bull 300

MANELLY, R B 1951 Blindness in newborn pigs Vet Rec. 63 398

MUHRER, M E. HOGAN, A G, AND BOGART, R 1912 A defect in the coagulation mechanism of swine blood Amer Jour Physiol 156 555

NELSON, P F 1911 Report of a case of true lateral hermaphroditism in Sus Anat Rec 80 1

NORBY, J E. 1929 An inherited skull defect in swine Jour Hered 20 229

NORDFELT, S 1911 Microphthalmia in swine Ann Agr Coll, Sweden 12 201

ROBERTS, F, AND MORRILL, C. C. 1944 Inheritance and histology of wattles in swine Jour Hered 35 149

ROSS, O B., PHILLIPS P H BOHSTEDT, G, AND CUNHA, T J 1944 Congenital malformations, syndactylism talipes and paralysis agitans of nutritional origin in swine Jour Anim Sci 3 406

RUNNELLS R A 1954 Animal Pathology 5th ed Iowa State University Press Ames Iowa p 51

SAUTER, J H., YOUNG, G A LEEDER, A J, AND KITCHELL, R J 1953 The experimental production of malformations and other abnormalities in fetal pigs by means of attenuated hog cholera virus Proc. Book, Amer Vet Med Assn 90th Ann Meet., p 146

SEIBOLD, H R., AND ROBERTS C S 1957 A microscopic congenital cerebellar anomaly in pigs. Jour Amer Vet. Med Assn 130 26

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Tumors, Intestinal Emphysema, and Fat Necrosis

Tumors

The best information on the prevalence of tumors in swine was gathered by Steiner and Bengston (1951) They obtained their data from the 1948–49 report of the Bureau of Animal Industry in the United States Department of Agriculture In that year more than 49 000 000 hogs were slaughtered in packing houses having federal meat in spection Of the 49 million animals the en tire carcasses of 1 247 were condemned for tumorous conditions This is only 0 003 per cent of the animals as contrasted to 0 063 per cent of the cattle which were con demned for the same reason In addition to these condemnations of entire carcasses it is estimated that parts of approximately 86 000 other pigs were condemned for tu mors

Tumors of swine then appear not to be very prevalent Steiner and Bengston point out however that these figures do not give the whole story with respect to the preva lence of swine tumors because only about two-thirds of the hogs are slaughtered in packing houses having federal inspection and these are healthy hogs The other third may and probably does contain more ani mals with tumors They emphasize also the fact that in 1918, 87 1 per cent of the ani

mal kill consisted of barrows and gilts un der one year of age They had not reached the tumor age

KINDS OF TUMORS IN SWINE

Day in 1907 began to give information on the prevalence of embryonal nephroma (Wilm s tumor) in swine slaughtered in Chicago Among 93 tumors in his swine collection 17 were of this type Feldman at the Mayo Foundation in 1933 had a col lection of 86 swine tumors among which were 16 embryonal nephromas In that same year Davis and his colleagues in list ing 26 swine tumors that came to them from Denver packing houses had 15 cm bryonal nephromas among 26 tumors Pre viously Kinsley in 1930 had reported one in a gilt In 1918 Plummer at Ottawa, Cana da had 2 in a group of 3 tumors he re ceived from abattoirs the previous year These data give considerable support to the current belief that this is the most pre valent tumor of swine

In Davis 1933 report 6 of his 26 swine tumors were of lymphoid origin In the same year Feldman classed 2, out of 77 in his collection as tumors of this type While the numbers are not large they do indicate why the lymphoblastomas are rated see n I in importance in tumors of p 5t.

lium predominate From the well developed capsule of connective tissue, strands of this tissue permeate the parenchyma of the tumor subdividing it into masses of various sizes and shapes The blood vessels of the stroma send capillaries into the parenchyma The parenchyma in some areas has the appearance of a fibrosarcoma, in others of an adenoma The cells of the sarcomatous areas are round, ovoid, or spindle The cells of the adenomatous portions seem to be attempting to form alveoli and irregular branching, blind tubules, and occasionally renal corpuscle like bodies The cells forming alveoli and tubules are cuboidal or columnar Those forming renal corpuscle like bodies are flat or cuboidal To complicate the pattern of the tumor, sinuses and cysts filled with blood or hyalin like material may be mixed with the other elements Occasionally smooth and striated muscle and, very rarely, cartilage and bone may be present also Some of the epithelial cells usually are in a state of mitosis

LYMPHOMA

The second most prevalent tumor among swine is the lymphoma, almost always the malignant lymphoma (lymphosarcoma lymphoblastoma)

Anatomical Location

This tumor arises in pre existing lymphoid tissue, usually in the lymph nodes Most, if not all, of the lymph nodes become affected Metastasis to other organs frequently occurs early It is surprising that the spleen is often exempt from involvement by this tumor In Feldman's collection of 16 such tumors from swine only 1 had splenic involvement On the other hand metastasis had occurred to the liver in 10 of the 16 hogs Metastasis may occur to other organs also In three-fourths of Feldman's cases the immature lymphocytes in the lymph nodes had become so numerous that they spilled over into the general circulation in numbers great enough to constitute a condition of lymphocytic leukemia

Kernkamp (1915) recorded a case in a pig only 55 days old which was allowed to

live to the 77th day All of the nodes were much enlarged

Gross Appearance

Involved lymph nodes are generally much enlarged and on the cut surface have the usual appearance of lymphoid tissue except that the volume of tissue is too great for the size of the capsule As a consequence, the cut surface bulges In other organs the metastasizing tumors may be discrete fleshy nodules usually fairly widespread, or the parenchyma of the organ may be diffusely infiltrated with the tumor cells This is generally what occurs in the liver

The enlarged lymph nodes compress neighboring organs or parts and result in a most varied combination of symptoms

Microscopic Appearance

Affected lymph nodes lose the usual anatomical pattern of peripheral sinuses cortical nodules and medullary cords with peritrabecular sinuses Proliferating lymphocytes take over the node so that the normal structure disappears and becomes one of a solid mass of immature large lymphocytes with hyperchromatic nuclei and numerous mitotic figures

The cells which form discrete tumorous nodules in other organs or infiltrate organs in a diffuse manner have the same appearance as those in the affected nodes Likewise, those tumor cells which appear in the circulating blood naturally have a resemblance to the parent cell

Plummer (1915) described a *reticulum cell sarcoma* of the bone marrow of an 8 month-old pig in Canada He saw only one-half of the dressed carcass but noted the presence of the tumor in the marrow of every bone including the coccygeal vertebrae The thoracic lymph nodes were enlarged The report is incomplete because the author did not have access to the whole carcass

Carcinomas are of little importance in swine In 1926 Feldman studied 15 swine tumors in a collection of 132 tumors of animals which he had received from various parts of the United States Nine of these were adenocarcinomas Later, in 1933, the same investigator listed 3 out of his own collection of 76 tumors as carcinomas Davis et al (1933) had 1 in 26

Melanomas and malignant melanomas seem to be of a little greater importance in swine than cancer Pickens in 1918 described one, Caylor and Schlotthauer (1926), 3, Feldman (1926), 3 out of 15 in his collection Davis et al , (1933), 1 out of 26 in their collection, and Jackson (1936) in South Africa 2 out of 6 in his collection

Other tumors of swine which have been described by various investigators are

Adenoma
Liver, 2   Feldman, 1935
Liver 1   Jackson, 1935
Fibroma
Skin, 1   Davis et al 1933
Fibrosarcoma
Pericardium, 1   Davis et al , 1933
Hemangioma
Skin, multiple, 1   Jackson, 1936
Ovary, cavernous 1   Davis et al , 1933
Myxoma, 1   Feldman 1926
Myxosarcoma 1   Feldman 1933
Papilloma
Glottis multiple, 1   Jackson 1936
Reticulum cell sarcoma
Bone marrow, 1   Plummer 1945

EMBRYONAL NEPHROMA

Feldman, who has made the most thorough study of the embryonal nephroma, states that it originates "as a consequence of some congenital mishap from multipotent, undifferentiated, nephrogenic cells" The variable structure of the tumor from different animals and even from different parts of the same tumor has caused pathologists to give it a variety of names such as adenomyosarcoma, sarcocarcinoma, sarcoadenoma, rhabdomyo adenosarcoma and adenosarcoma The variation in structure is explainable on the grounds that the tumor is congenital, it arises from the cells of

primitive nephrogenic tissue As in most fetal rest tumors the rate of growth and differentiation of the originating cells is variable and one or more types of cells may outgrow and displace other types so that the end results in a number of cases may not have much resemblance Since the tumor has its origin in the embryo and develops from nephrogenic tissue, the simplest and most satisfactory way to name it is to call it an embryonal nephroma This tumor seldom affects the health of the animal because it is usually unilateral and most hogs having it are slaughtered young

Anatomical Location

As stated previously the embryonal nephroma is the most common tumor of swine It is usually located in the kidney but if not in the organ itself, it is located nearby, either anteriorly or posteriorly The incidence is practically the same for both sexes Since in most instances the tumor is discovered at slaughter, age incidence means little This is the more true because the tumor at some stage of development must be present even at birth

Gross Appearance

The tumor generally originates in the cortex of the kidney and more often at either pole It does not occur at the hilus It may consist of a single or a few grayish white nodules projecting from the surface of the kidney or, at the other exteme, consist of a large grayish white lobulated mass which displaces most of the organ Day (1907) saw one which weighed 60 pounds Large tumors may become cystic, the cysts being filled with blood tinged urine or blood tinged purulent fluid The larger tumors, which are also the older ones are practically always well encapsulated This suggests that metastasis should be rare and that is usually the case If it does occur the lung is the favored site for secondaries

Microscopic Appearance

Basically the embryonal nephroma is a heterogeneous mixture of tissue elements among which connective tissue and epithe

Fat Necrosis

Fat necrosis in swine, like intestinal emphysema, is practically never recognized during the life of the animal It is only at necropsy examination in a packing house in practice, or at a veterinary college that areas of death of adipose tissue either externally or internally are ever seen Even the external form may not be seen then because the subcutaneous fat is still covered by the skin

Gross Appearance

Areas of fat necrosis occur in normal adipose tissue as sharply defined yellowish white to chalky white opaque foci a millimeter or so in diameter up to irregularly shaped patches several centimeters in size They are firm like soap If they become calcified, they have a gritty feel

Microscopic Appearance

When adipose tissue dies a chemical change occurs Lipase splits it into fatty acids and glycerine The glycerine being soluble is absorbed and removed from the area The fatty acids remain at the site and assume the form of acicular crystals which are laid down in a hit and miss fashion Calcium in the lymph which bathes the dead tissue combines with the fatty acids to form calcium soap With the usual histological stain calcium soap has a bluish pink granular or homogeneous appearance within the adipose tissue cells Calcification may progress to completion The affected cells then become entirely blue or have a margin of blue with a bluish pink center

Cause

Experimentally internal fat necrosis has been produced by traumatizing the pancreas and the pancreatic duct When this is done the proteolytic and lipolytic enzymes escape into the peritoneal cavity The protease probably attacks the cell membrane and permits the lipase to enter and decompose the fat It is presumed then that spontaneous fat necrosis in pigs must be preceded by pancreatic damage of some kind which permits pancreatic juice to escape into the body cavity where it attacks the peritoneal fat and causes fat necrosis

This explanation does not seem satisfactory for the external or subcutaneous form of the condition It has been found that connective tissue contains both a protease and a lipase When connective tissue is traumatized these enzymes are released and probably account for the subcutaneous fat necrosis which occurs in swine when they have been bruised some time prior to slaughter

In China it was suggested that the source of the enzyme which causes fat necrosis might be a lipase present in vegetable matter fed the hogs In Missouri however in an experiment in which hogs were fed large amounts of soybeans and peanuts both rich in lipase, no fat necrosis occurred

In areas of southern United States where the kidney worm (Stephanurus dentatus) of swine is prevalent necrosis of the abdominal fat occurs frequently It is presumed that the necrosis is in some way associated with this form of parasitism Perhaps stray worms invade the pancreas or pancreatic duct

REFERENCES

Tumors

CARLOR H D, AND SCHLOTTHAUER C F 1926 Melano epithelioma of the ear of the swine and its cultural experiments Arch. Path. and Lab. Med. 2 313.
CHAMBERS F. 1936 The incidence of cancer in animals an analysis Vet Rec 48 1 3.
DAVIS, C. L, LITTER, R B, AND SHELTON J F 1933 Neoplasms encountered in federally inspected establishments in Denver Jour Amer Vet Med Assn 3 229

characterized by small white, fleshy nodules in the spleen and sometimes in the liver and lymph nodes made up of a variety of cells, the principal ones being lymphocytes, eosinophils, and a few giant cells The condition resembles Hodgkin's disease in man The cause is unknown Because of its lack of progressiveness it is probably not a true tumor

MELANOMA

Melanomas of swine are generally malignant and are primary in the skin They occur in young pigs are often multiple, and metastasize early If the affected animal lives long enough, the melanomas become generalized

Pickens (1918) described malignant melanomas which covered several areas of the skin on the anterior half of the body The pig was a 6 to 8 week-old Duroc Caylor and Schlotthauer (1926) observed this tumor in the skin of 3 young Durocs The primary tumor in one was in the flank region Metastases were found in the lungs liver, kidneys, and in some lymph nodes

The malignant melanomas studied by Davis and by Feldman were likewise primary in the skin

The histological appearance of melanosarcoma is familiar to every one who has had a course in general pathology, so a description of it will be omitted here

Intestinal Emphysema

Intestinal emphysema of swine is a condition found only at necropsy This must not be construed to mean that the change occurs after death because it does not It occurs in apparently healthy living hogs but is not detected until the animal is slaughtered While discovery is usually made at the slaughter house, it occasionally is seen also in hogs that are necropsied in the field or in diagnostic laboratories

Gross Appearance

Gas filled vesicles varying in size up to about 2 cm in diameter occur in the lymphatics of the mesentery near the place of attachment to the intestine, and in the lymphatics of the wall of the intestine, principally in the jejunum and ileum The vesicles are solitary or multiple At times they are present in conglomerate masses or patches bulging from the surface of the serosa At first the wall of each vesicle is clear or translucent Later the wall becomes red from congestion Vesicles in the mucosa and submucosa may project into the lumen of the intestine

Microscopic Appearance

The lymphatics in all layers of the intestinal wall are distended as a result of the gas The wall of each vesicle in a lymphatic consists of an accumulation of macrophages many of which have formed multinucleated foreign body giant cells containing vacuoles where gas was present The surrounding connective tissue is infiltrated with lymphocytes and numerous eosinophils

Cause

The cause of intestinal emphysema is not known In Germany it was believed to be due to feeding hogs whey It has been suggested that it may be the result of excessive intestinal fermentation Biester and associates (1936) observed it in pigs in Iowa that were reared on a ration consisting to large extent of polished rice Pigs raised on polished rice supplemented with corn and skim milk had no intestinal emphysema but those fed polished rice with protein added had the emphysema the same as those fed the rice alone No conclusions were drawn as to the exact cause of the condition

Surgery

DAY L. E 1907 Embryonal adenosarcoma of the kidney of swine U.S.D.A Bur Anim Ind
    24th Ann Rept Washington D C
FELDMAN W H 1926 *A study of the tumor incidence in the lower animals* Amer Jour Path
    2 545
—— 1928 A study of the histopathology of the so called adenosarcoma of swine Amer Jour
    Path 4 125
—— 1930 The so called lymphoid hyperplasias of animals Jour Amer Vet Med Assn 30 294
—— 1932 Neoplasms of Domesticated Animals W B Saunders Co Philadelphia
—— 1935 Metastasizing hepatoma in a hog (*Sus scrofa*) Amer Jour Cancer 27 111
FORBUS W D AND DAVIS C L 1946 *A chronic granulomatous disease of swine with striking
    resemblance to Hodgkin s disease* Amer Jour Path 22 35
JACKSON C 1936 The incidence and pathology of tumors of domesticated animals in South
    Africa Onderstepoort Jour Vet. Sci and Anim Ind 6 3
KERNKAMP H C H 1945 Lymphoblastoma in a pig Jour Amer Vet Med Assn 106 155
KINSLEY A T 1930 An interesting case of adenosarcoma of a gilt Vet Med 25 362
PICKENS E M 1918 Generalized melanos s in a pig Jour Amer Vet Med Assn 52 707
PLUMMER P J G 1945 Reticulum cell sarcoma of the bone marrow of a pig Canad Jour
    Comp Med 9 254
—— 1948 A survey of twenty four tumors collected from animals slaughtered for food Canad
    Jour Comp Med 12 180
STEINER P E. AND BENGSTON J S 1951 Research and economic aspects of tumors in food pro
    ducing animals Cancer 4 1113

Intestinal Emphysema

BIESTER H E EVELETH D F AND YAMASHIRO Y 1936 Intestinal emphysema of swine Jour
    Amer Vet Med. Assn 41 714

Fat Necrosis

FARR C E 1923 Ischemic fat necrosis Ann Surg 77 513
NEAL M P 1916 Fat necrosis studies VI The effect of feeding lipase containing vegetable
    seed on the production of fat necrosis Arch Path 41 37

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Preparation for Operation

INSTRUMENT STERILIZATION

Proper sterilization of instruments plays an integral part in the aseptic chain of surgery To insure proper sterilization the instruments must first be thoroughly cleaned Blood pus, tissue, and other debris protect pathogenic organisms from being killed by the various sterilization procedures Thorough cleaning is greatly facilitated by various commercial detergents These agents not only facilitate the loosening of foreign material but also have little detrimental effect on the instruments themselves

used Arlein and Walters (1913) state that boiling instruments in water for 30 minutes will destroy bacterial vegetative forms while boiling in alkaline water requires only half that time Addition of 2 per cent sodium carbonate or 0 1 per cent sodium hydroxide not only reduces sterilization time but also decreases the corrosive action It is necessary to note that, although boiling instruments in water for 30 minutes will destroy bacterial vegetative forms it is not sufficient to destroy most bacterial spores In struments sterilized by this method should be thoroughly cleaned, unlocked and completely covered by water Sterilization time

humid atmosphere   This is because of their small lung capacity and the layer of fat around their bodies which slows the elimination of heat   If it is necessary that they be restrained under such conditions then it is advisable to handle the pigs in the early morning hours and before the temperature rises   Always be sure that there is adequate ventilation of the quarters in which they are confined   This is important in cool as well as in hot weather   When a large group of pigs is being handled, it is advantageous to have a smaller pen connected with the main pen   Then a small group (not more than 25 or 30 weanling pigs) can be moved into relatively small quarters where they can be caught more easily   It also keeps the main herd from becoming excited and prevents piling and overcrowding which may be

There are many ways of restraining pigs   Those described here are commonly used in the north central region of the United States   In general it can be said that for the person who is skilled in the handling of pigs their restraint seems easy while to one who is not so skillful it appears quite difficult   A person holding a smaller pig off the ground by its fore or hind legs or holding a large pig by its fore legs with the rump resting on the ground, can hold the pig steadier if he leans or rests against some solid object   The holder can use the side of the pen the wall or a couple of bales of straw   This is not so important in handling a small number of pigs as it is a large group when the holder's back is apt to become fatigued

Handling Suckling Pigs

ting instruments as it has no effect on the temper of the cutting edges The corrosive action of many of the agents used is eliminated by addition of sodium carbonate or sodium nitrite The bactericidal efficiency of most of the agents used in chemical sterilization increases proportionally with the temperature of the solution According to McCulloch (1946), solutions maintained at 100 to 110° F give a more reliable sterilization than those maintained at room temperature Spaulding (1939) tested various agents used in chemical sterilization and found that none of the germicides tested killed *Clostridium tetani* spores in less than 18 hours Instruments should remain in chemical sterilizing solutions for at least 30 minutes to insure destruction of bacterial vegetative forms and for more than 18 hours to insure destruction of bacterial spores The presence of foreign material such as blood, pus, feces, and soap on contaminated instruments not only lengthens sterilization time but also will inactivate cationic detergents such as benzalkonium chloride (Zephiran) Some widely used solutions in chemical sterilization are as follows

Alcohol Formaldehyde solution

Formaldehyde 37%	130 ml
Potassium nitrite	0 15 gm
Sodium hydroxide	0 012 gm
Ethyl alcohol 95%	q s 1,000 ml

Benzalkonium chloride solution (Zephiran)

Benzalkonium chloride 10%	40 ml
Sodium nitrite	40 gm
Sodium carbonate	40 gm
Distilled water	q s 1 gal

Benzalkonium chloride Alcohol Formaldehyde solution

Benzalkonium chloride 10%	14 ml
Isopropyl alcohol	900 ml
Methyl alcohol 70%	72 ml
Formaldehyde 37%	114 ml
Sodium nitrite	12 gm
Sodium carbonate	12 gm
Distilled water	1 800 ml

Sterilization of surgical shrouds, towels, and sponges can be effected by either the steam or dry heat method Allam (1918) recommends that a pressure of 15 pounds per square inch at 250° F for 30 minutes be used for sterilization of textile material If a pressure cooker is used, the materials should be wrapped in heavy paper as this affords a more rapid drying following sterilization The water may also be removed from the pressure cooker following sterilization to facilitate drying If additional drying is necessary, McCulloch (1946) recommends placing the pack in a drying oven at 230°F McCulloch (1946) also states that overnight baking at a temperature of 244 to 252° F is sufficient for sterilization of clean shrouds

Surgical gloves can be sterilized in chemical sterilizing solutions or in an autoclave If chemical solutions are used, one should remember that the gloves must be free from foreign material and must be kept in solution for at least 30 minutes

HANDLING AND RESTRAINT OF PIGS

Pigs are difficult to drive or put into pens The use of panels or gates greatly facilitates driving them into pens or smaller enclosures A stick or stockman's cane placed near the face of the pig where it can be seen is useful in turning the animal The pig can be urged forward by tapping or pushing on the rump, however, care must be taken not to bruise the pig Slappers" (a strip of belting attached to a stick) are much better for driving a group of pigs and they do not bruise the pig

When pigs confined in a hog house or enclosed compound become disturbed or excited they will try to escape by forcing their way through any suitable opening where they see any light If a number of pigs are penned together for the purpose of catching, they will frequently crowd against the sides or walls of the pen to avoid being caught The time given to examining the wall or fence to make certain that they cannot break out is usually time well spent When pigs are penned, plenty of good dry bedding should be provided This holds down the dust, cleans and dries the feet of the pigs, and makes for easier handling

Pigs, especially fat hogs, cannot withstand much exertion in a hot and/or

stretching out much the same as shown in Figure 45.3 for weanling pigs. If the pig can rest its nose on the ground, it cannot be held steady by this method. The pig will use its nose as a brace on the ground and by struggling will keep throwing the holder off balance. Shoats and the larger pigs are caught by standing behind the animal and reaching over its back to grasp both of the front legs, then quickly lifting up in order to set the pig on its rump (Fig. 45.4). Again the front legs are brought back by the ears to prevent the pig from biting the handler's hands.

For castration without anesthesia a larger pig of this group can be thrown on its side by reaching under the pig and grasping the front and hind legs on the opposite side. The legs are jerked toward the handler to throw the pig on its side. The handler then quickly grasps the two upper legs and stepping behind the pig places his knee in the cervical region. The operator then carefully places his knee in the flank region to help restrain the pig while castrating. Needless to say, the pig is placed on the left side for a right-handed operator and on the right side for a left-handed one.

Troughs can be used for restraining the larger shoats for vaccination. The shoat is grasped by the front legs and tipped up on its rump at which time a second man grabs the hind legs and the pig is placed in a trough. The trough either is built to be
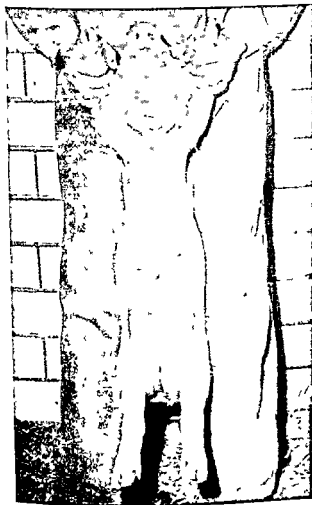
FIG. 45.3 — Restraint of a weanling pig by the front legs for vaccination or other procedures. The front feet are drawn back to the region of the ears and pressed against the side of the head. This steadies the head and exposes the axillary space. Note that the thumbs are out of reach of the pig's mouth.
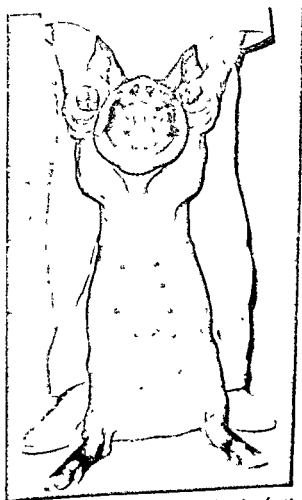
FIG. 45 4 — Restraining a shoat by the front legs.

held by the front legs in the manner used for weanling pigs shown in Figure 45.3

Handling Weanling Pigs

Weanling pigs can be caught by the hind legs and held stretched out as shown in Figure 45 2. The knees are used to restrain the thorax of the pig and prevent wriggling movements Pigs of this size can also be restrained by holding the front legs and using the knees to limit movement of the body (Fig. 45.3). However, one must be careful to bring the front legs back by the ears so that the hands, especially the thumbs, are out of reach of the pig's mouth or the holder may be bitten

For more extensive procedures where anesthesia is used, the pig can be held or suspended by the tarsal joint. If general anesthesia is used, this can be given first and then the pig suspended. Pigs that are not held during the procedure may be suspended in a trough or board leaned against the wall. A table can also be used. A small rope or a chain is placed around the leg above the tarsal or hock joint and then an

additional half-hitch should be placed around the leg distal to the tarsal joint This is because of the ease with which ropes or chains can slip over the tarsal joint. Even when general anesthesia is used it is advisable to restrain the front legs by means of a rope or chain with an additional half-hitch around the leg. Some veterinarians have a bar in a trough under which the head slips, others put a rope or chain through the mouth, which assists in restraining the head.

Handling Shoats

The means used to restrain shoats will depend on the size and strength of the pig as compared to the size and strength of the person handling the pig. The shoats should be crowded into a small enclosure to facilitate catching. The smaller ones can be caught by the hind legs and restrained by

FIG. 45.1 — Restraint of a suckling pig for castration.
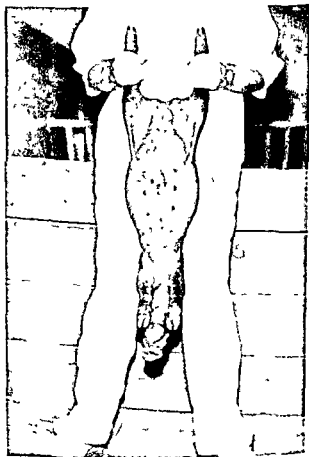
FIG. 45.2 — Restraint of a weanling pig by the hind legs for castration or vaccination. The knees are used to prevent wriggling movements.

height as the pig s snout   Then either a piece of pipe from 12 to 16 inches long with rings welded on each end or a singletree is placed between the pig s hind legs A piece of obstetrical chain or rope is threaded through each ring and around the hind leg on that side of the pig   This chain or rope is attached to a block and tackle which is anchored to a post at a height of about 6 feet and the block and tackle tightened to stretch the boar A modification of this method is to attach the block and tackle to a beam above the pig and lift the rear end of the pig off the ground This modi fication of the method can be used to raise larger pigs for hernial operations Some times a block and tackle is attached to each hind leg and then each block and tackle is attached to the beam at a different point so as to pull the hind legs apart when tight ened

Bullard (1956) gives boars a general anesthetic agent until they fall   Then a rope is tied to the lower hind leg, brought through the mouth, then passed around the upper hind leg, and tightened This pulls the hind legs forward and out of the field of operation for castration Kendrick (1954) fastens a rope 4 feet long around the left front leg with a slip knot The rope is then passed to the right front leg and anchored to it with a half hitch The hind legs are treated in the same manner An assistant holds the rope and, when the pig falls from anesthesia the rope is tight ened to turn the pig on its back Then the rope from the front legs is passed between the hind legs and under the rope attached to them The rope from the hind legs is passed under the rope on the front legs and the ropes pulled tight This pulls all four legs together

For cesarean section a pig can be stretched between two posts by means of ropes on the front and hind legs Some men use a block and tackle to aid in stretching out the pigs The sides of the pen can also be used for anchoring the legs The upper hind leg may need to be released when the wound is sutured Another way to restrain pigs for cesarean section is to tie them to a gate The wooden gate should have open

ings between boards through which ropes can be passed All four legs are tied to the gate Ropes are passed around the pig s body just anterior to the tuber coxae and the hind leg Another rope can be passed over the thorax, but care should be taken to see that it is not tight enough to inter fere with respiration The third loop of rope is placed over the cervical region After the pig is restrained the gate can be raised to a height convenient to the sur geon by placing it on bales of straw or blocks

ANESTHESIA

Local Anesthesia

Infiltration is the most common method of producing local anesthesia in pigs It consists of injecting an anesthetic agent in to the tissues of the area to be anesthetized In this way the nerve endings and the nerve fibers in the area are anesthetized Since only a local area is involved this type of anesthesia can be used on patients which would be poor risks if general anesthesia were used Local anesthesia is also used for minor surgical procedures however it is not entirely satisfactory in pigs A re strained pig tends to continue squealing and struggling even if pain in the surgical area has been abolished For that reason local anesthesia is often combined with a general anesthetic agent which is given for sedation There is not space in this chap ter to describe the technique of local infil tration in the various areas In general, it can be said of the various parts of the body wall that the skin is most sensitive Almost as sensitive are the serous coverings of the body cavities such as the parietal perito neum

Procaine hydrochloride in a 2 per cent solution and hexylcaine hydrochloride (Cy claine) in a 1 per cent solution have proved satisfactory for local anesthesia in the pig

Epidural Anesthesia

Epidural anesthesia is produced by in serting a hypodermic needle between two vertebrae and depositing an anesthetic agent in the epidural space of the vertebral

set on the ground or is elevated. The front legs of the pig are pulled forward and held back of, or at the level of, the ears to prevent the pig's biting the holder. The hind legs are pulled in the opposite direction.

Squeeze chutes of various types are used to restrain pigs. They are placed at the end of a corridor or at a door. Most of them have an opening much like a stanchion through which the pig puts his head in trying to escape. The stanchion is then closed on the pig's neck. There is a self closing stanchion on the market which is popular in some areas.

Various types of mechanical hog holders are also used on the larger pigs of this group. Their canine teeth or tusks are not developed to the point where they will prevent a rope placed over the snout from slipping over the teeth, thus releasing the pig. Devices making use of a cable or wire to grasp the end of the snout will hold these pigs. The cable or wire is passed through a pipe, and a loop is formed on the end. This loop is placed over the snout back of the canine teeth. Then the end of the pipe is pressed against the top of the snout and the wire or cable tightened. The pig is held by pressing downward against the snout with the pipe, tightening the loop around the snout at the same time. Mechanical devices which are round or octag-

onal in shape and of the proper size to fit over the end of the snout are sometimes used. They are forced backward in the mouth until they are behind the canine teeth and then pulled forward. This places upward pressure on the inside of the mouth and downward pressure on the outside of the snout. In other words, the instrument is prying on the snout. The mechanical advantage of these devices is so great that fractures of the bones forming the snout have resulted from their use.

Pigs can be cast by using various types of casting harnesses. One of these is shown in Figure 45 5. The pig is held with a hog holder of the type which has a cable passed through a pipe. A short piece of chain is passed over the mechanical holder and looped over the snout. Another piece of chain is fastened above the tarsal joint. A rope is passed from the chain around the snout through the chain fastened to the leg and back through a ring on the chain around the snout. The rope is then quickly tightened, pulling in the direction indicated in Figure 45.5, while at the same time the handler's knee is thrust into the pig's flank to throw it off balance. The rope is tied to the hind leg after the pig has been cast.

Another method of restraining large hogs is the one devised by Alcorn of Iowa (1953) or a modification of it. The boar is first snubbed to a solid post at about the same

FIG. 45 5 — Use of a modified casting harness for casting a pig.

regular and abdominal in type The sensi
tivity of the skin now begins to disappear
It disappears first in the abdominal region
and then in other regions of the body The
skin of the back and the coronary band are
the last areas of the skin to lose their sensi
tivity When these signs are present, the pig
is in the plane of the surgical stage of anes
thesia where major surgery can be per
formed During the recovery stage the sen
sitivity returns first in the coronary band
and in the skin of the back The sensitivity
of the rest of the skin gradually returns as
do the other signs in the reverse order of
their disappearance

ANESTHETIC AGENTS

Thiopental Sodium, U S P (Pentothal sodium, Thiopentone sodium, Leopental)

Thiopental is a short acting thiobarbitu
rate In pigs Muhrer (1950) reported
anesthesia lasting from 4 to 70 minutes.
Jacobsen (1955) reported recovery in
about 30 minutes

Thiopental sodium is available commer
cially in ampules as a crystalline powder
buffered with anhydrous sodium carbonate
It is readily soluble in water but is unstable
in aqueous solution or when the powder
is exposed to moisture of any type including
that in the air From his studies on the
stability of thiopental sodium, Robinson
(1947) recommended that any unused solu
tion should be kept no longer than 3 days
at room temperatures of 64 to 71° F (18 to
22° C) or no longer than 7 days at 41 to
42° F (5 to 6° C.) If turbidity occurs be
fore these expiration times the solution
should be discarded The solution still has
anesthetic action but the concentration is
decreased making it difficult to estimate
dosage Thus solutions of this drug are
generally mixed immediately before ad
ministration

The instability of the aqueous solution
of thiopental sodium makes it desirable to
estimate rather closely the dosage of the
drug needed so that the proper amount will
be available The estimated dosage varies
somewhat with different authors as does the
speed of injection

Muhrer (1950) calculated the minimal
surgical dosage on 86 pigs of various weights
(Table 45 1) Diehl and Weischeider
(1956) reported on the use of thiopental
sodium on 380 pigs Their dosage ranged
from 4 5 mg/lb (0 9 ml of a 5 per cent
solution per 10 lb of body weight) in pigs
weighing slightly over 100 lb to 13 mg/lb
(0 86 ml of a 5 per cent solution per 10 lb
of body weight) in pigs weighing 550 lb
Jacobsen (1955) gives the amount of thio
pental sodium to have in the syringe to in
sure having enough rather than the
amount used For large pigs he had 9
mg/lb in the syringe, and for small pigs
he had 13 6 mg/lb available

The intraperitoneal route of administra
tion is little used for thiopental sodium
Clover (1955) reported inconsistent results
when the drug was given intraperitoneally
while Jacobsen (1955) found it difficult to
control the level of anesthesia and also
found that the time elapsing between injec
tion and anesthesia was uncertain

The technique of administering thio
pental sodium varies with the different
authors. Muhrer (1950) and Clover (1950)
recommend giving one half of the calcu
lated dose fairly rapidly so as to minimize

canal The solution bathes the spinal
nerves which have emerged from the dura
and produces anesthesia in the areas sup
plied wholly by the nerves anesthetized
Epidural anesthesia is used for anesthesia of
the perineal, inguinal, and abdominal re
gions

Location of the anatomical position for
insertion of the needle for epidural
anesthesia is more difficult in swine than
in most other species This is due to the
layer of fat which covers the dorsal spines
of the vertebrae The site for injection used
in swine is the lumbosacral space (Frank,
1953 Wright 1939) If the site cannot be
palpated Frank (1953) recommends draw
ing an imaginary line connecting the ante
rior borders of the wings of the ilia The
junction of this line with the dorsal mid
line crosses over the center of the last lum
bar vertebra (Wright 1939) Frank (1953)
inserts the needle about 2½inches posterior
to landmark located by drawing the imagi
nary lines in an average size pig The
needle is directed ventrally and anteriorly
at an angle of about 45° Wright (1939)
inserts the needle directly posterior to the
dorsal spine of the last lumbar vertebra
found by use of the imaginary line, and
directs the needle in a ventral and poste
rior direction to enter the vertebral canal
The dosage that Frank (1953) recommends
is 1 ml of a 2 per cent solution of procaine
hydrochloride to 10 pounds of body weight
If the surgical area is posterior to the peri
toneal cavity, the dosage can be reduced

General Anesthesia

In the production of general anesthesia
the anesthetic agent is carried by the blood
stream into contact with the various com
ponents of the nervous system The physi
cal and chemical characteristics of these
agents determine which route or routes can
be used for their administration The
routes which can be used to introduce
anesthetic agents into the blood stream are
intravenous inhalation, intraperitoneal, in
trapleural, and gastrointestinal Intrave
nous, inhalation and intraperitoneal
routes are most widely used for general
anesthesia in swine

Each route of administration has its ad
vantages and disadvantages which may vary
according to the agent used When the in
travenous route is used, the drug is injected
into either the anterior vena cava or one
of the ear veins For the technique of enter
ing these veins the reader is referred to
Chapter 48 The intravenous route re
quires the best restraint of the three
methods as the needle must be kept in the
vein for a period of time The inhalation
route requires good restraint but more
movement can be tolerated than with the
intravenous route The intraperitoneal in
jection of anesthetic agents in the smaller
pigs is not difficult The pig is held by
the hind legs with the head down and ab
domen toward the person making the in
jection as shown in Figure 45 3 The in
jection is made on the lateroventral part of
the abdomen above the level of the umbili
cus This is to avoid the bladder in females
and the bladder and prepuce in males The
onset of anesthesia varies and the depth of
anesthesia is inconsistent when anesthetic
agents are administered intraperitoneally

The signs of the various stages of
anesthesia which are seen in the pig vary
with the route of administration If the in
travenous route is used, they vary with the
speed of administration When the intra
peritoneal route is used, the struggling and
excitement that often occur during the
second or delirium stage of anesthesia are
seldom seen In the administration of
anesthetic agents by the intravenous route
it is best to pass through the stage of de
lirium rather rapidly and then continue
slowly to effect In most instances it is
difficult to observe the eye of the pig for
signs of anesthesia because of its location in
the head and the shielding position of the
large ears Thus more attention is paid to
other signs The first sign of leaving the
stage of delirium and entering the begin
ning of plane 1 of the third stage or the
stage of surgical anesthesia in a standing
pig is swaying on the hind legs This indi
cates the beginning of motor paralysis
which extends to the front legs and the rest
of the body As the stage of surgical anes
thesia is entered, the respiration becomes

under restraint   The operative site was then prepared and infiltrated with a local anesthetic agent   By the time the local infiltration was completed the sow was recovering from the general anesthesia   Depression of fetal respiration was not observed

Thiamylal sodium is a good anesthetic for short surgical procedures on pigs   It is relatively safe and has a smooth period of recovery

Pentobarbital Sodium, U S P (Nembutal sodium, Halatal, Somnopentyl, Narcoren Narcoven)

Pentobarbital sodium is classified as a short acting member of the barbiturate group   It is longer acting than either thiopental sodium or thiamylal sodium   Commercial solutions which are very stable are readily available in approximately 6 per cent solutions   Nestman (1954) reported anesthesia of 45 to 60 minutes duration and recovery in 5 to 6 hours when administered intravenously   Kernkamp (1939) reported recovery from anesthesia in 60 to 90 minutes   The difference in recovery time could be explained by the difference in size of the pigs and the depth of anesthesia obtained   Dosages recommended by the different authors are given in Table 45 2

The margin of safety with pentobarbital sodium decreases with increase in weight of the pig   It is a fairly safe anesthetic agent in small pigs but the margin of safety decreases greatly from 100 lb up to the higher weights   The procedure generally followed in large swine is to inject enough of the drug to cause a standing pig to fall

The pig is then restrained by tying its legs   If necessary the general anesthesia is supplemented with local infiltration of a suitable agent   In other words pentobarbital sodium is not given to effect in large pigs   For smaller sows not more than 10 ml is injected   larger sows should not receive more than 12 to ml   depending on their size   Likewise large boars should not receive more than 18 20 ml (Bullard 1956) of the solution   If pentobarbital sodium is given to produce surgical anesthesia in these large pigs   the danger of respiratory failure is great and the recovery period may be 10 to 12 hours or longer   Some of the animals may die during the long recovery period

The rate of injection for the administration of pentobarbital sodium should be about 5 ml per minute according to Kernkamp (1939)   Wright (1939) recommended injecting over a period of 3 to 4 minutes

When administered intraperitoneally pentobarbital sodium is given at the rate of 13 mg/lb or 1 ml of a 6 2 per cent solution per 5 lb of body weight (Kernkamp 1939)   Recovery from anesthesia is seldom less than 1 hour from the time of onset when this route is used   according to Kernkamp (1939)

Chloral Hydrate, U S P

Chloral hydrate used intravenously produces anesthesia lasting from 30 to 60 minutes (Slatter 1918 Bajez 1935)   The recovery period is about 3 hours (Klarenbeek and Hartog 1938)   When the intraperitoneal route is used the time between injection and the onset of surgical anesthesia is about 15 minutes   the duration of

TABLE 45.2
RECOMMENDED INTRAVENOUS DOSAGE OF PENTOBARBITAL SODIUM TO PRODUCE ANESTHESIA IN PIGS

Author	Size of Pig (lb)	Dosage (mg/lb)	Ml of 5% Solution per 10 lb of body weight (approximately) (ml)
Kernkamp (1956)	80 110	8 6	1 3
Wright (1939)	up to 200	13 0	2 0
Wright (1939)	more than 200	not over 9 0	not over 1 4
Adam and Churchill (1946)	100 150	6 0-9 0	0 9-1 4

the excitement period and then give the remainder slowly Administration is stopped when surgical anesthesia is obtained Jacobsen (1955), as stated above, dilutes the estimated dose of thiopental sodium with about 20 ml of water He injects 1 ml and waits 1 minute to observe the reaction to the drug Then if no idiosyncrasy is noted he injects 1 ml every ½ minute until the pig falls He waits 1 minute before resuming injection and then continues at the same rate until surgical anesthesia is obtained

The effect of thiopental sodium on the circulatory and respiratory systems of pigs varies with the dosage and the rate of administration Jacobsen (1955) found that the drug had a marked depressant action on the respiratory system which was directly proportional to the size of the dose and the rapidity of injection A fast injection resulted in a fall in blood pressure Muhrer (1950) found that respiration was affected before the pulse rate and that respiratory failure preceded cardiac failure

Dreisbach and Snyder (1943) found that thiopental sodium decreased fetal respiratory movements in rabbits at about the same rate as pentobarbital sodium, but because of the shorter duration of anesthesia in the dam the effect was shorter in duration

Thiopental sodium is a good anesthetic for use in surgical procedures of short duration More of the drug can be administered for a longer period of anesthesia, but since it localizes in the adipose tissue (Brodie *et al*, 1952), the recovery period will be long when a large amount of the drug is given It is a relatively safe anesthetic agent since respiration fails first and, if artificial respiration is instituted at that point, the losses will be few However, it is not as convenient to use as some other anesthetics because of its instability in solution and expense as compared to other barbiturates

Thiamylal Sodium

Thiamylal sodium is a member of the thiobarbiturate group and is classified as an ultra short acting anesthetic agent Dunne and Benbrook (1954) reported that in swine it produced anesthesia which lasted approximately 10 to 12 minutes When clinical doses were used, the animals were able to stand in 30 to 40 minutes

The drug, according to Dunne and Benbrook (1954), had no apparent effect on the circulatory system and moderate effect on the respiratory system when clinical doses were used

Thiamylal sodium is available commercially as a powder buffered with sodium carbonate in sealed ampules A 4 per cent solution is the concentration usually administered to swine Lumb and Armistead (1952) found that a 4 per cent solution retained its potency up to 14 days at room temperature Innes (1956) reported that a 4 per cent stock solution retained its potency for at least 7 days It appears that a sterile solution of 4 per cent thiamylal sodium solution can safely be stored for at least a week and perhaps 2 weeks at room temperature

Thiamylal sodium can be administered by either the intravenous or the intraperitoneal route Dunne and Benbrook (1954) found the dosage to be the same by both routes They reported the dosage to be 8 mg/lb or 1 ml of a 4 per cent solution per 5 lb of body weight This dosage was based on anesthetizing over 200 pigs weighing from 25 to 70 lb They administered the drug intravenously by injecting rapidly to within 1 ml of the estimated anesthetic dose The effect was noted and the remainder given slowly until surgical anesthesia was obtained When the intraperitoneal route was used, they injected the drug and if there was no sign of anesthesia in 5 minutes a second injection was made using one third of the original dose If this did not produce the necessary results, supplementary amounts were given by the intravenous route

Higbee (1956) used thiamylal sodium in cesarean sections He injected 0 5 gm rapidly via the intravenous route to sows weighing from 300 to 500 lb When the sow dropped the legs were tied as described

larities, many of which occurred during the induction of anesthesia. The full signifi cance of these irregularities is not known at the present time. However, it indicates the potential danger if the administration is not carefully regulated to prevent over dosage.

Boyd and Kernkamp (1940) used chloro form in the castration of boars. The pig was snubbed to a post by means of a rope placed around the snout. A section of burlap was wound lightly around the jaws and fastened with a small rope or bandage. Then the drop method of administering chloroform was used. Kendrick (1954) placed a feed bag over the snout instead of the burlap. Some veterinarians in the north central region of the United States have found that a mask made of heavy wool fashioned to fit over the pig's snout gives more satisfactory results.

According to Boyd and Kernkamp (1940), the first sign of anesthesia is the swaying of the body followed by the animal's falling on its side. The mask is then removed unless the pig needs more chloroform. The ordinary boar requires about 60 ml (2 oz) of chloroform.

There are some precautions which should be taken in the use of chloroform on pigs. The pig should be restrained so as to mini mize movement during the excitement and delirium stage. Chloroform should not be given outside on a windy day as it is diffi cult to achieve a high enough concentration in the pig for anesthesia. A pig is some times anesthetized but does not fall be cause its feet are braced in such a way that the rope on the snout holds the pig up. Thus any pig that stops struggling during the administration of chloroform should be given a shove on the rear quarters to see if it will fall. Such a pig, if not tested, could

PREPARATION OF THE OPERATIVE SITE

The bacterial flora of the skin is com posed of two types, resident and transient (Price 1938). The resident bacteria are firmly attached to the skin and are also located in the hair follicles, sebaceous glands and sweat glands. Removal of all resident bacteria is almost impossible. Transient bacteria vary tremendously in number and species depending on the animal's environment. They are found in greatest numbers about the feet, axillary inguinal and perineal regions. They may include both saprophytic and pathogenic species. In the pig Armistead (1956) was able to isolate Staphylococcus aureus from only one animal in four from a previously prepared and disinfected skin area. How ever one should not turn out pigs with open surgical wounds into a grossly contaminated hog lot or pen. The pigs should be turned into a clean pen with new bedding or a clean grass lot.

Following the restraint of the hog the

anesthesia about 90 minutes, and the recovery period about 2¼ hours according to Klarenbeek and Hartog (1938).

The average intravenous dose recommended by different authors is given in Table 45.3.

Slatter administered chloral hydrate solution intravenously by means of a simple gravity unit until the pig fell and then continued until he obtained the desired effect. Prugelhof (1954) carefully estimated the dosage and then slowly injected the drug with a syringe until the desired stage of anesthesia was reached. Bajez (1953) injected chloral hydrate solution in fractional doses. He injected 5 ml. of the 40–50 per cent solution, waited 5 seconds, and then injected 5 ml. more. He continued at this rate until the pig lost control of its hind legs and fell. If necessary, he continued administration at the same rate until the proper stage of anesthesia had been reached. Bajez (1953) has given this drug to 800 pigs in this manner without a fatality.

When the intraperitoneal route of administration is employed, a 5 per cent solution of chloral hydrate is commonly used Klarenbeek and Hartog (1938) starved the pig for 24 hours before administering the anesthetic agent The dosage used was 113–151 mg/lb (2 2–3.0 ml. of a 5 per cent solution per pound of body weight). They seemed to prefer the larger dosage most of the time. Hassler (1952) used 113 mg/lb, or 2.2 ml. of a 5 per cent solution, as the dose intraperitoneally. There is some question as to the amount of inflammation caused by the chloral hydrate solutions in the peritoneal cavity. Klarenbeek and Hartog (1938) stated that the injection caused

a mild inflammation which was of no consequence. Hassler (1952) stated that 10 of 84 pigs injected in this manner died, most often a month or more following surgery. He was not able to examine any of the pigs after death but believed that death was due to peritonitis produced by the chloral hydrate.

Chloral hydrate given intravenously to large pigs appears to be a fairly safe anesthetic agent. When the slower methods of administration with the less concentrated solutions are used, the animal must be well restrained.

The intraperitoneal route is the one most used in small pigs The anesthesia is fairly satisfactory if the dosage is correctly calculated. However, the possibility of peritonitis detracts from its usefulness.

Chloroform, U.S.P.

Chloroform is the drug most widely used for inhalation anesthesia of swine in this country. A few practitioners use ether, but it requires a special mask unless induction is made with some other agent. Chloroform has been used for short surgical procedures such as the castration of boars and in longer procedures such as cesarean sections in sows. Chloroform has the advantage of a short period of excitement and quick recovery. However, the administration must be carefully watched as it affects the heart before it affects the respiratory center and may cause death by various cardiac irregularities, including ventricular fibrillation. Stowe and Hammond (1954) ran electrocardiograms on 18 pigs given chloroform as a general anesthetic agent. Twelve of these pigs showed cardiac irregu-

TABLE 45 3
RECOMMENDED INTRAVENOUS DOSAGE OF CHLORAL HYDRATE SOLUTIONS TO PRODUCE ANESTHESIA IN PIGS

Author	Size of Pig	Mg Per Pound of Body Weight	Solution	Ml Per 10 lb of Body Weight
			(%)	
Klarenbeek and Hartog (1938)	large	68–79	20	3 4–3 95
Slatter (1948).	large	66	33	2 0
Bajez (1953)	220 lb	45 4	40–50	0.9–1 1
Bajez (1953) . . .	660–880 lb	30 3	40–50	0 6–0 8
Prügelhof (1954).	220 lb.	56 8	50	1 1

NESTMAN, H   1954 Kurze Betrachtung zur Narkose des Schweines mit Narcoren Tierärztl
    Umschau 9 431
PRICE, P B   1938 New studies in surgical bacteriology and surgical technique Jour Amer Med
    Assn 111 1993
——— 1950 The meaning of bacteriostasis bacterial effect and rate of disinfection Ann New
    York Acad Sci 53 76
——— 1951 Disinfection of skin Drug Standards 19 161
PRUGELHOF, Γ   1954 Die intravenose Chloralhydratnarkose in der tierarztlichen Praxis Wien
    tierärztl Monatschr 41 627
ROBINSON, M H   1947 Deteriorations of solutions of pentothal sodium Anesthesiology 8 166
SLATTER, E E   1948 Anesthesia in swine No Amer Vet 29 157
SPAULDING E H   1939 Studies on chemical sterilization of surgical instruments Surg Gynec
    and Obst 68 738
STOWE, C M   AND HAMMOND, P B   1951 Unpublished data
WRIGHT, J G   1939 Observations on the use of nembutal as a general anaesthetic in the pig the
    technique of partial hepatectomy in the pig spinal (epidural) anaesthesia in the pig
    Jour Comp Path and Therap 52 1

gical area This tends to wash the bacteria away from the incision The alcohol is then allowed to air dry, and a 2 per cent tincture of iodine is applied in a similar manner

PREPARATION OF THE SURGEON

One of the weaker links in the chain of asepsis is the preparation of the surgeon The fingernails should be trimmed short and the nails and cuticles cleaned The hands and arms should be scrubbed briskly with soap and rinsed with clean water for 7 minutes A 3 minute scrub with tincture of Zephiran or 70 per cent alcohol follows Many practitioners use the 3 to 5 minute scrub with hexachlorophene-detergent Price (1950) has shown that if hexachloro

phene detergent is used several times a day for 4 or more consecutive days, the resident bacterial flora is reduced to 5 per cent of its original number The low flora will be maintained as long as the hexachlorophene detergent is used faithfully However, as soon as it is discontinued, the flora again will become re established A single scrub with hexachlorophene detergent has no more rapid germicidal effect than ordinary soap

Following preparation of the surgeon's hands and arms, sterile gloves are donned Surgical gloves not only protect the animal from bacterial contamination from the surgeon's hands but also protect the surgeon when performing a grossly contaminating procedure

REFERENCES

Alcorn, H A 1953 Cited by E. R Frank (1953)
Allam, M W 1948 Sterilization aseptic technique in veterinary practice Jour Amer Vet Med Assn 112 338
——— And Churchill E A 1946 Pentothal sodium anesthesia in swine and goats Jour Amer Vet. Med Assn 109 355
Arlein M S And Walters C W 1943 Aseptic technique in veterinary surgery Jour Amer Vet. Med Assn 102 41
Armistead W W 1946 The preoperative sterilization of skin No Amer Vet. 37 675
Bajez E. 1953 Zur intravenosen Chloralhydratnarkose beim Schwein Wien tierarztl Monatschr 40 282
Boyd W L. And Kernkamp H C H 1910 Castration of boars under chloroform anesthesia No Amer Vet 21 287
Brodie B B Bernstein E. And Mark L. C. 1952 The role of body fat in limiting the duration of action of thiopental Jour Pharm and Exper Therap 105 421
Bullard J F 1956 Personal communication
Clover R H 1955 Some experiences of thiopentone sodium as an anesthetic in the pig Vet Rec. 67 354
Diibl, H And Weischeider A 1956 Die Pentothalnarkose beim Schwein Wien tierarztl Mona tschr 43 31
Dreisbach R., And Snyder F F 1943 The effect on fetus of pentobarbital sodium and pentothal sodium Jour Pharm and Exper Therap 79 250
Dunne, H W., and Benbrook S C 1951 A note on suntal sodium anesthesia in swine Jour Amer Vet. Med Assn 124 19
Frank E.R 1953 Veterinary Surgery Burgess Publ Co Minneapolis
Higbee, J M 1956 Personal communication
Hässler, L. 1952 Några synpunkter på intraabdominal narkos hos yngre svin Nord Vet Med 4 593
Innes D C. 1956 Personal communication
Jacobsen, P T 1955 Leopental Danske Dyrl Medlembl 38 70
Kendrick J W 1954 Chloroform anesthesia for castrating boars Vet Med 49 501
Kernkamp, H C H 1939 Narcosis and anesthesia in swine produced by pentobarbital sodium Jour Amer Vet Med Assn 91 207
——— 1946 Personal communication
Klarenbek A., And Hartog, J H 1938 Der heutige Stand der Anaesthesie Anwendung Proc 13th Int Vet Cong Part 1 553
Lumb W A., and Armistead W W 1952. Surital sodium anesthesia in small animal surgery No Amer Vet. 33 175
McCulloch L. C. 1946 The role of disinfection in veterinary medicine II Sterilization of surgical instruments Jour Amer Vet. Med Assn 108 242
Meurer, M E. 1940 Restraint of swine with pentothal sodium Jour Amer Vet Med Assn 97 293

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Operations Involving the Testicle and Inguinal Canal

There probably are no areas of the animal body in which more surgery is performed than in the scrotal and inguinal regions This is particularly true with swine due mainly to the large numbers castrated In addition, there are many that are affected with pathological conditions requiring surgery, such as cryptorchidism scirrhous cord, and scrotal hernia

The description of these operations will be confined almost entirely to the actual operative procedures The causes pre surgical preparation, anesthesia, and after care will be discussed only briefly as these topics are considered in another chapter

CASTRATION

The object of the operation is to destroy the function of the testicle It may be ac complished in two different ways In one method the blood supply to the testicle is actually destroyed with a resulting atrophy, this method is not practical to apply to swine The other is surgical removal

Every operator has some particular technique of his own choice which he uses during the course of the operation He has in mind such things as size and loca tion of the initial incision, the extent to which it penetrates the tissues, and the length of the cord The amount of tunic removed will also vary Likewise, the age of the animal has an important bearing on the procedure that he will follow

With our present day methods of swine management, practically all of our swine are castrated as small pigs from a few days of age up to the age of weaning It is ad visable to examine these small pigs for the presence of existing hernias and crypt orchidism When castrating a large number at one time, those afflicted with these con ditions should be put aside and operated separately Thus the routine for castrating large numbers will not be disrupted

Restraint when castrating small pigs is not a serious problem although it is im portant to have them properly confined Methods for restraint are described in de tail in Chapter 45

The incision is made over the testicle on the somewhat distended scrotum This distension is produced by placing the hand in front of the scrotum and then exerting a pushing back movement while pinching with the thumb and forefinger This raises the scrotum slightly and facili tates making the incision The incision is carried through all structures into the testicular tissue This causes the testicle to pop out through the incision in the tunic After discarding the scalpel, the testicle is grasped and pulled or twisted in such a manner that the cord will separate (Figs 46 1 and 46 2)

This method frequently does not remove any appreciable amount of tunic along with the testicle, in small pigs it is not

and with a downward thrust the cord is severed. No further dissection is done. In most cases healing is uneventful. However, if some unforeseen condition should occur, such as prolapse or excessive hemorrhage, it would be much easier to correct these conditions if the boar were under general anesthesia and in a recumbent position.

After castration, swine should be examined frequently, especially in fly season. Repellents should be used when necessary Adequate shelter should be provided. Specific individual treatment is seldom required.

CRYPTORCHID CASTRATION

Cryptorchidism is defined as a developmental defect in which the testicles fail to descend, and remain within the abdomen or inguinal canal. True cryptorchidism, therefore, is the condition in which both testicles are undescended, while monorchidism applies to a unilateral retention or absence.

These terms are used rather loosely, and to most people cryptorchidism means one or both testicles retained, and cryptorchid castration likewise refers to castration which

FIG. 46.1 — Testicles forced backward cause scrotum to become distended prior to making incision.

FIG. 46.3 — Skin incision only, shows testicle covered with tunic.

FIG. 46.2 — Testicle exposed by direct incision as result of reflection of tunic.

FIG. 46.4 — Low incisions, obscured from view when pig is in standing position.

so important to remove this tunic. If removed, it is dissected with scissors. After the testicle is removed, one often observes a small incision. In small pigs it is usually not necessary to enlarge it as it would be in older animals. It is better, however, to have a fairly large incision to provide good drainage, even in small pigs. It is easily enlarged with scissors.

Another procedure that is very satisfactory starts as described above, but deviates in that the initial incision is made down only to the outer or parietal tunic. At this point, with the thumb and fingers one can easily force the testicle, with its outer tunic still intact, through the skin incision. If one grasps the testicle and applies moderate traction, at the same time pressing down on the scrotum with the other hand, the testicle will be elevated sufficiently so that the entire cord can be crushed and severed (Fig. 46.3). This so-called "covered type" of castration might seem to one who has not done it to take too much time. With some practice, however, it is as rapid as the other method and eliminates the after-dissection of the tunic since the tunic is removed in its entirety with the testicle. The incision may be enlarged as before.

In the case of barrows for show purposes, some owners prefer to have their pigs castrated "low" to avoid visible scars. The only point necessary to consider here is the location of the incision. All other procedures are the same. The incision is usually made slightly anterior to the point where the scrotum is reflected onto the abdominal wall (Fig. 46.4). In this position, the scars will be between the legs and will not be seen. If pigs are castrated properly, even high on the scrotum, the scars that remain are rather difficult to see and do not distract from the appearance in any manner.

When castrating large boars, one of the most important points to consider is restraint. It is often said that good anesthesia is one of the best types of restraint, and this certainly is true when castrating large boars. With the proper use and application

of general anesthesia, one can carry out carefully and completely all the necessary steps in this or any other operation. Anesthetized animals make it much easier to do top-grade surgery. Here the veterinarian has the opportunity to demonstrate his skill and training (see Chapter 45).

The skin of the scrotum is very thick and tough, making it difficult to incise. If incisions are bilateral, it is often advantageous to make a small puncture wound through the tough scrotum. A probe-pointed bistoury is inserted, and the skin is incised from the inside out. The midline incision is much more desirable as the skin is not nearly so thick, and a regular incision can be made easily (Fig. 46.5).

The tunic in large boars is closely adherent to the scrotum over a considerable area. It takes some dissection at this point to free the testicle with its tunic intact if one is to do the closed operation (Fig. 46.6). After isolating these structures, the cord can be followed easily towards the internal ring, at which point the cord is crushed and severed with the emasculator (Fig. 46.7). After both testicles have been removed, the septum is dissected, and the incision enlarged, if necessary to provide drainage.

The most commonly practiced method of castration in boars is one in which the initial incision is carried all the way into the testicular tissue at one time. It should be noted that, if this method is used, there will be a reflection of the heavy tunic from the testicle. Removal of the tunic is accomplished by dissection with scissors. However, it is usually more difficult to remove it by this technique than when it is removed with the testicle in the covered operation.

The operation may be greatly simplified. One method is to snub the boar to the side of a fence. A supporting rope is tied to the top of the fence. It is passed under the boar just in front of the hind legs, then brought back up, and again tied to the fence top. It is adjusted so that it prevents the animal from going down in the hind quarters. Two bold incisions are made over each side and deep enough to enter the testicular tissue. Each testicle in turn is grasped securely,

be grasped with forceps, nicked and opened with scissors The retained testicle is found frequently in this area However, it may require a careful and prolonged search with the fingers If one is successful, he first must be able to recognize all anatomical structures such as the gubernaculum testis epididymus, and cord

The testicle, after being located, is brought through the ring The cord is ligated, after which a crushing forceps or clamp may be applied The cord is then separated In small pigs it is usually sufficient merely to crush and separate the cord A sufficient number of mattress or interrupted sutures are placed in the ring to close it properly Any one of several suture materials may be used, such as linen, cotton silk, nylon, gut, or cable wire Umbilical tape is also a very reliable material It is more often used in larger hogs

The remainder of the abdominal wall may be closed by using a subcuticular suture, in order to obliterate any cavity formation This is accomplished by using a continuous suture which brings the subcutaneous layers into apposition The next step is to suture the skin Interrupted or mattress sutures work very well A through and through including the subcutaneous tissues and skin may be substituted satisfactorily after the ring has been closed

The methods described for closing the abdominal wall will result in an early healing process Some operators prefer to do no more suturing after the inguinal ring has been closed If this is all that is done, the area becomes secondarily infected, and some subcutaneous sloughing occurs It is usually slight and is soon followed by granulations Healing eventually takes place, and the final results are satisfactory Advocates of this technique argue that the extra scar tissue formed as a result of infection gives added support to the area This particular statement is especially true in the case of umbilical hernias

Johnston (1956) describes a technique for cryptorchidectomy in pigs which is very similar to the method just described It differs mainly in the location of the abdominal incision He starts it about an inch below the external inguinal ring and extends it downward for approximately 1 to 1½ inches

Another location of approach that has been employed rather frequently by the author is in the paralumbar fossa or flank A standard laparotomy is done, making all incisions through the various structures in the same plane This is done to facilitate the closing of the incision after the removal of the testicle

Frequently, if the various muscle layers are separated parallel to their fibers, it may be difficult to remove the testicle as such a method does not ordinarily allow a very

FIG 46 7 — Tunic and testicle removed, leaving clean operative wound requiring no further dissection

may be unilateral or bilateral. The correct medical term, cryptorchidectomy, is seldom used, especially in this country.

As with routine castration, cryptorchid castration is more satisfactorily performed when the pigs are relatively small. Pigs with an average weight of 30 to 10 pounds are of an ideal size for operation.

The surgical approach may be made in different anatomical locations, such as the inguinal region or the paralumbar fossa, or flank. Often it is necessary to operate only one side when the flank approach is elected, even if both testicles are retained.

The operative area selected will depend upon the choice of the operator. The operation can be done satisfactorily under local infiltration anesthesia.

If the inguinal approach is selected, an incision 2 to 3 inches long is made over the internal ring An incision of this length is usually sufficient to expose the ring, especially after some blunt dissection has been done.

At this stage a careful examination should be made to determine if the testicle is inguinal in position. If not, the peritoneum is ruptured with the fingers, or it may
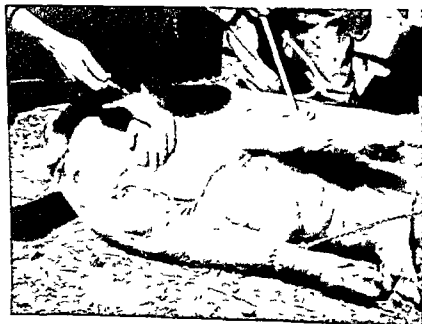
FIG. 46.5 — Midline incision, castration of large boar.

FIG 46 6 — Exposed testicle and tunic in covered operation

be grasped with forceps, nicked and opened with scissors The retained testicle is found frequently in this area However it may require a careful and prolonged search with the fingers If one is successful he first must be able to recognize all anatomical structures such as the gubernaculum testis epididymus, and cord

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Frequently if the various muscle layers are separated parallel to their fibers, it may be difficult to remove the testicle as such a method does not ordinarily allow a very

FIG 46 7 — Tunic and testicle removed leaving clean operative wound requiring no further dissection

large opening in the wall Also, with this technique, the peritoneum is more likely to be incised in an irregular manner, and it is difficult to bring the incision into proper apposition

The retained testicle often will be palpated immediately through the incision when it is grasped with the fingers or forceps. If it is not located immediately, the middle and index fingers are moved in a circular manner By doing this, the testicle is usually found By lifting up and supporting the pig under the lower flank, the search is made more easily It is seldom necessary to insert the entire hand, especially in small pigs In bigger pigs, the incision may be enlarged to allow passage of the entire hand, and a much more complete search can be made

If there is a bilateral involvement, the opposite testicle often can be located and removed, but with somewhat more difficulty Occasionally it may be necessary to do a bilateral laparotomy

If one operates many pigs, he will occasionally find an animal in which it is nearly impossible to locate the testicles In these few cases, if one feels he is not justified in continuing, he may close the incisions in the usual manner However, if he is determined to find them, the only recourse left is to make a large midline incision and take advantage of visual inspection It might be well to mention that a careful examination for operative scars should always be made before starting the operation

After the testicle is located and exteriorized, the cord should be ligated or clamped (Fig 46 8) Again, in small pigs, clamping and crushing is all that is necessary To complete the operation, a few properly spaced sutures are inserted

REMOVAL OF SCIRRHOUS CORD

Scirrhous cord refers to a hard tumor like mass which develops on the end of the spermatic cord after castration It is characterized by an enlargement of the scrotum At some point on the surface of this enlargement there is at least one small fistulous opening, from which there is a continuous discharge of a small amount of a thin purulent exudate (Fig 46 9)

There are extensive subcutaneous adhesions in the affected parts, and especially in the region of the fistulous areas On dissection, a fine tortuous tract is seen to lead to a small central core of infection and necrosis This is completely surrounded by a very dense, firm, thick connective tissue capsule

The operation is best performed with the pig in dorsal recumbency under general or local infiltration anesthesia The latter is satisfactory in most cases

Two cutaneous incisions are made in such a way that they isolate an elliptical island of skin, which should include the fistulous opening This isolated section should be as large as is practically possible The reason for this is obvious, in that there are extensive adhesions between the skin and the underlying tissues This procedure will minimize the amount of dissection between these structures

FIG 46 8 — Flank approach for cryptorchid castration, testicle exteriorized and clamped

By holding this area with forceps considerable traction may be applied which will facilitate the separation of the remainder of the scirrhous mass from the adjacent tissues (Fig 46 10) This separation is accomplished by careful snipping and cutting It is continued until all the dense adhesions have been separated

Finally a point is reached where the fingers may be employed for blunt dissection At this stage there is natural cleavage between the cord and the surrounding tissues The cord is easily separated down to the inguinal ring (Fig 46 11) By using one's fingers in this area the danger of injuring the penis is greatly reduced Such an injury might easily occur if cutting instruments were used

The cord is now transfixed and ligated as close to the internal ring as possible It is then clamped and severed At this point the ring should be carefully examined for evidence of enlargement If enlargement is found a few mattress sutures will usually eliminate the possibility of prolapse

Usually moderate hemorrhage is encountered during the operation due to the large area of tissue exposed If much hemorrhage does occur a sterile gauze pack may be inserted over the ring for a period of 24 hours Often packing is not necessary

FIG   46 9 — Scirrhous   cord with f stulous open ng appearing in center of enlargement

FIG   46 10 — Ell pt cal island w th f stulous open ng n teeth of forceps
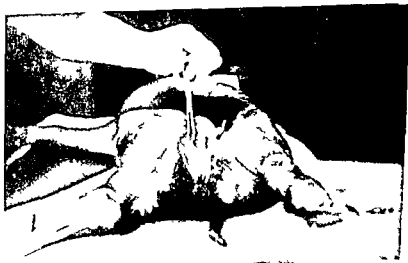
and a liberal application of an astringent dusting powder will suffice A fly repellent should be used if pigs are operated during the fly season

Considerable local infection and superficial necrosis develop These are followed by normal granulations covering the entire exposed area Healing is usually uneventful

SCROTAL HERNIA

Scrotal hernia refers to the passage of intestines, usually the small one, into the inguinal canal (inguinal hernia), then by extension into the scrotum when it becomes a scrotal hernia

It is characterized by an enlargement which is soft and easily manipulated (Fig 16 12) It may occur bilaterally If reducible as most of them are, the contents can be forced into the abdominal cavity

Pigs should be operated when small Those weighing on an average, 30 to 40 pounds, are ideal size They may be suspended by the hind legs However, it is more satisfactory if they are placed on some supporting structure (see Chapter 45) It is also advisable to have the hind quarters elevated If not suspended a pig should be placed on its back secured in this position by tying ropes on all four legs, and then tied to the supporting structure Any method of restraint that can be worked out

will do, so long as the hind legs are held somewhat apart to expose the inguinal region

Satisfactory anesthesia can be obtained by injecting a few cubic centimeters of a local anesthetic subcutaneously over the internal ring in the area where the incision is to be made The size and location of the incision are similar to that described for cryptorchid castration After the incision has been made, the fingers are used to separate the subcutaneous tissue sufficiently to expose the spermatic cord with its parietal tunic intact. Through this tunic the intestines are usually visible

The scrotum is now grasped with one hand while the index finger of the other hand is bent into a hooked position and forced gently around the cord to separate it from the adjacent tissues (Fig 46 13)

A stripping motion using the thumb and index finger forces the intestines into the abdominal cavity Now firmly grip the cord With a pulling and prying motion while still holding the scrotum, the peritoneal covering of the testicle will separate from it The scrotum is now released Next slide the hand up the cord and grasp the testicle It is then rotated with a wrist motion sufficiently strong to put a solid spiral twist in the cord (Fig 46 14) This automatically forces the intestines into the abdomen This step is necessary since fre

FIG 46 11 — Scirrhous mass isolated with enlarged edematous cord extending into inguinal ring at point where ligation is made

FIG 46 12 — Scrotal hernia with extensive intestinal displacement Area over lower enlargement is usual location for operative incision

FIG 46 13 — Finger hooked around isolated cord for applying traction in separating tunic from scrotum

FIG 46 14 — Twisted cord forces intestines into abdominal cavity, exposing ring
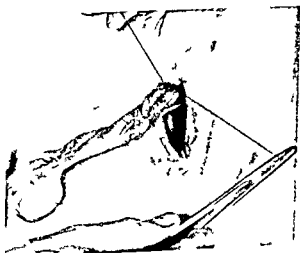
FIG 46 15 — Ligated cord confines intestines, Cord is severed and stump sutured into ring gives added support to area

quently the intestines will work back through the canal during the manipulation of disengaging the tunic from the scrotum

A pair of forceps could be placed low on the cord just as soon as the stripping has been accomplished This would hold the intestines in the abdominal cavity while the tunic was being separated from the scrotum This is usually not done however, because the forceps would prevent complete twisting of the cord

During this procedure one should be extremely careful to keep from tearing the outer tunic It is not serious if it happens but it is much easier to reduce the hernia if the intestines are still retained

The cord, with its tunic, is transfixed and ligated as close to the ring as possible (Fig 16 15) The entire cord is crushed with forceps and severed The ligature used for this should be left sufficiently long so that the cord stump can be tied easily into the ring This is accomplished by passing a suture through both sides of the ring and tying with a square or triple throw knot

In some cases where the ring is unusually large a few additional mattress or interrupted sutures are necessary While applying these sutures, the hind leg on the involved side should be loosened to relieve tension so that closure may be made more secure An antiseptic dusting powder may be applied if one terminates his operation at this point If this is done, healing will be prolonged due to secondary infection

It is better, after closing the ring either to use a suture which incorporates the subcutaneous tissue and skin, or to apply separate sutures to each By this procedure primary healing will occur in the minimum period of time However, in the majority of cases, after securing the cord stump into the ring, a through and through suture is all that is necessary for healing to take place A dusting powder on the incision completes the operation

It is not considered good surgery to use catgut sutures in the skin However, if about a No 1 medium chromic gut is used as suture material, the sutures need not be removed This is a practical consideration since in most situations pigs that are operated are turned out with the others and are usually not examined again

If a non reducible hernia is encountered it requires much more careful dissection to free the adhesions Frequently it becomes necessary to enlarge the ring to reduce the hernia Careful closure in these cases is extremely important. The remainder of the operation is the same as for a reducible hernia

It is hoped that the author's descriptions of these operations will provide a working basis for those who wish to perform them Naturally some modifications will be employed by any given operator

Any of the operations described can be accomplished much more quickly if some of the details are omitted, but, if an operation is done in a slipshod manner, the end results may be disastrous Let us as trained veterinarians, be meticulous in our operative procedures

REFERENCE

Johnson R W 1956 A technique for cryptorchidectomy in pigs Vet Rec. 68 277

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Operations Involving the Female Genital Tract

CESAREAN SECTION

Dystocia in the sow is of frequent occurrence and presents many difficulties in determining the exact cause of the trouble If the genital tract is too small to permit the passage of the fetuses it is also too small to make a manual examination In some cases a few fetuses may have been born without difficulty, but examination may reveal an unusually large fetus that is too large to pass through the pelvis It will be impossible to know if this is the last fetus or whether there are a number of other fetuses still in the uterus In some cases the patient has had a normal number of fetuses but is still uneasy as though there were one or more fetuses in the uterus Deciding whether it is indicated to attempt an embryotomy to use manual extraction, or to perform a cesarean section in certain cases is important if the patient is to live

A cesarean section will be uniformly successful in those cases that are operated within a few hours after parturition is first observed Also the amount of injury that has occurred to the soft tissues of the genital tract in attempts to relieve the dystocia may be a determining factor If repeated attempts have been made to extract a fetus with little regard for asepsis infection has usually been introduced into the damaged tissue A cesarean section will be unfavorable if delayed more than 18

hours after the beginning of parturition The operator who has had a great deal of experience is a close observer and has unusual intuition is usually most successful in choosing those cases in which a cesarean section will result in a high percentage of recoveries

Anatomy of the Genital Organs of the Sow

The ovaries are usually located along the lateral margins of the pelvic inlet in the young animal but in the older animal they may be found farther forward In the sow the ovaries are lobulated due to the projection of the follicles and corpora lutea above the surface of the ovary The Fallopian tubes are long but not as tortuous as in the mare and have a rather large abdominal opening Some of the unusual features of the uterus are the short body about 2 inches long and the horns which are very long and flexuous In the non pregnant animal the horns may be 4 or 5 feet long and may become 10 or 12 feet long during pregnancy The neck is rather long and continuous with the vagina which is 4 or 5 inches long The termination of the genital tract is the vulva which is about 3 inches in length

Preparation for Operation

If possible the operation should be performed in the hospital where the proper facilities are available for it We prefer to

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confine the animal on the table in lateral recumbency with the right side uppermost For those operators that prefer to operate in the median line, the patient will need to be confined in dorsal recumbency The operative area on the side extends from the last rib to the tuber coxae and below the ends of the transverse processes of the lumbar vertebrae The skin is shaved and prepared in the usual manner for an aseptic operation

Anesthesia for the operation may be obtained by a number of different methods If an experienced anesthetist is available, one of the volatile anesthetics may be used Chloroform is very effective in bringing the patient to the stage of maintenance Then ether may be used to keep the patient in the stage of maintenance while the operation is being performed If chloroform is used during the entire operation, its effect must be closely watched as it is a powerful anesthetic

When the patient is toxic, probably the safest procedure is to use a local anesthetic. A 1 or 2 per cent solution of procaine hydrochloride may be used to infiltrate the skin along the proposed line of incision After the skin incision is made, the muscles and peritoneum may be infiltrated on the same line as the skin incision

Another method of producing anesthesia is to give 10 cc of nembutal intravenously to control the struggling of the patient Then anesthesia of the tissues is obtained by infiltrating along the proposed line of incision with a local anesthetic If a general anesthetic is given intravenously for complete general anesthesia, the fetuses receive too much of the anesthetic and do not recover from its action

Epidural anesthesia may be used, particularly in the small gilt. The needle should be inserted into the vertebral canal between the last lumbar vertebra and the first sacral vertebra through the lumbosacral space The point to insert the needle on the median line is located by drawing an imaginary line across the anterior borders of the wings of the ilia Then approximately 2½ inches posterior to this line a needle is inserted through the skin A 4 inch, 18 gauge needle is directed in a downward and forward direction at about a 45° angle Depending upon the thickness of the fat over the area, the needle will need to be inserted a distance of 3 to 4 inches As the needle is inserted through the tissues, the point may strike the posterior edge of the spine of the last lumbar vertebra, which may be used as a guide to the lumbosacral space The needle may strike the edge of the bone before passing through the lumbosacral space A 2 per cent solution of procaine hydrochloride is used for epidural anesthesia For a 225 lb gilt, 20 cc are used and 25 cc for a 250 lb gilt The tail becomes relaxed in a few minutes if the anesthetic solution has been injected into the epidural space After the anesthetic solution has been injected, it is important not to incline the body anteriorly for the anesthetic solution may gravitate too far forward and affect some of the vital centers

Operation

The incision through the skin is 7 or 8 inches long, depending upon the size of the patient If the incision is too small, the operator may find it is difficult to bring the fetus enclosed in the uterus through the incision When the uterine wall is friable, it may be ruptured if the opening in the abdominal wall is too small The operative area is covered with a sterile rubber shroud which has an opening approximately 3 inches long in its center The rubber shroud is preferred in the sow as there is so much material from the uterus after a few fetuses have been removed that it is difficult to keep any other type of shroud clean The incision through the muscles is made the same as the skin incision When the transversus muscle is incised, there may be a thick layer of fat between it and the peritoneum It will facilitate the operative procedure to remove some of this fat in the wound before

incising the peritoneum The peritoneum is picked up with forceps and nicked with a scalpel and the incision finished with scissors A hand is introduced into the abdominal cavity to find one horn of the uterus Then a fetus enclosed in the uterus is brought out through the incision and this procedure is continued until the entire horn is on the outside

Whenever a cesarean section is performed without bringing all of the horn out through the incision, the operator may later find that he failed to remove all of the fetuses The operator will have to decide how many incisions will need to be made through the uterine wall to remove the fetuses In some cases one incision will be sufficient while in another case it may be necessary to make an incision over each fetus. When the uterine wall is incised the incision is made on the dorsal surface of the horn and large enough so the fetus may be easily removed Several fetuses may be removed through the one incision by inserting the hand and grasping a fetus It is usually necessary to work the uterine wall from the hand and the fetus like a glove as the fetus is removed If the fetal membranes are easily detached, they are removed, otherwise they are left to be expelled in a normal manner When infection is present in the uterus, various products that are available for combating infections may be inserted before the incision is closed The incision or incisions in the horn may be closed with one continuous Connell suture using No 1 chromic catgut Before returning the horn to the abdominal cavity, it is moistened with a mild antiseptic solution The other horn is now brought to the outside and the fetuses removed in the same manner A hand should be inserted through one of the incisions in the uterus to the pelvis to extract any fetus that may be present As soon as the incisions have been sutured and tissues moistened, the second uterine horn is returned to the abdominal cavity If the uterine muscles seem to be atonic 2 or 3 cc of pituitary extract may be in

jected into the blood stream The needle may be inserted into the vessels of the broad ligament or the ear vein Almost immediately the muscles of the uterus will start to contract rather vigorously

The incision in the peritoneum is closed with one continuous suture using No 1 chromic catgut One continuous suture using No 1 chromic catgut may be used to close the incision in the transversus internal, and external oblique muscles The edges of the skin incision are united with interrupted sutures placed about 1 inch apart

It is remarkable how little aftercare is necessary following cesarean section in the sow When there is any evidence of infection it may be treated with antibiotics or the sulfonamides The skin sutures should be removed in 10 days

HYSTERECTOMY

When the uterus is examined after performing the laparotomy incision, and the fetuses are emphysematous and the uterine wall is friable and possibly necrotic, the only chance to save the patient is to perform a hysterectomy However, when the uterus is removed in these cases, the mortality will be high due primarily to the shock of removing the large volume of tissue Also there will have been absorption of toxins and poisons from the uterus and the patient will be toxic During the operation the tissues must be handled very carefully as the uterine wall is easily ruptured One horn is brought through the incision to the outside as this procedure facilitates the ligation of the blood vessels One ligature is used to ligate the utero-ovarian artery and one the uterine artery in the broad ligaments First, the attachment of the ovary is divided and then the broad ligament It is best to divide the uterus as far posterior as possible, usually at the junction of the body and neck Before dividing the tissues, a ligature should be placed around the body of the uterus to prevent leakage from the uterus Before dividing the tissues forceps are fastened

so as to close the neck of the uterus, the tissues are divided between the ligature and forceps. The blood vessels to the other horn and ovary are ligated, the tissues divided, and the horn removed. Sutures are now inserted in the neck so as to invert the end into the vagina. The incision in the abdominal wall is closed in the usual manner.

Prolapse of the Vagina

A prolapse of a portion of the vulva and vaginal wall is sometimes observed in the sow. When this occurs, it is usually due to some irritation or injury to these tissues, and it has stimulated excessive straining. A soothing preparation may be applied to the irritated areas and the prolapsed tissue returned to its normal position. To retain the tissues in position, sutures are inserted in the tough skin lateral to the lips of the vulva on one side and then over to the other side. The sutures are pulled tight enough so the lips of the vulva are brought in close opposition. Also they are tied so they cannot spread apart. These are left in position for 10 days.

Oophorectomy in the Sow

The removal of the ovaries from the gilt or sow is an operation that has never been popular in this country. In Europe it is reported that thousands of females have been operated because they seem to fat ten more quickly.

The operative area is on the left side between the last rib and the tuber coxae. The operative area is prepared for an aseptic operation and the skin infiltrated along the proposed line of incision. The incision in the skin should be 4 or 5 inches long or just long enough to permit the introduction of the hand. The muscles are divided in the direction of their fibers and the fingers are pushed through the peritoneum. The hand is passed back to the pelvis and a search made for the ovaries. Failing to find the ovaries, one of the horns is picked up and followed to the ovary. The spaying shears are inserted along the arm to the ovary and it is removed, the other ovary is located and it is brought out. The skin incision is closed with interrupted sutures by bringing the edges together.

REFERENCES

Sisson, S 1953 The Anatomy of the Domestic Animals 4th ed Revised by James D Grossman W B Saunders Co , Philadelphia

Wooldridge, G H 1934 Encyclopaedia of Veterinary Medicine, Surgery and Obstetrics Vol II Veterinary Surgery and Obstetrics, 2nd ed Oxford University Press London

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Miscellaneous Operations

REPAIR OF UMBILICAL HERNIA

Several methods of operative procedure have been described and advocated for the repair of umbilical hernia in swine Most methods described are usually successful the techniques being sound in principle, the variations depending on the operator's experience with the particular method with which he has had the most success (Bullard, 1950, Frank, 1942, Guard, 1948) Regardless of the individual operative technique used, adherence to proper surgical principles and an understanding of wound repair are necessary for continuing success in surgical repair The handling of umbilical hernias should fit into the swine management program, the preferred time of operation is on young pigs following vaccination and weaning, or when the pigs are 8 to 10 weeks of age Swine with umbilical hernias should not be used for breeding purposes

The aim of operative surgery is the correction of the defect with a rapid repair of the tissues involved Assuming proper techniques, the other main factor in fluencing repair is bacterial contamination Ideally, all surgery should be done using sterile procedures throughout, and un doubtedly better results can be obtained if we adhere to strict asepsis and sterile tech niques Such strict asepsis is difficult to obtain in much of our farm surgery, but every effort should be made to come as

close to asepsis as possible Surgical packs can be prepared beforehand by autoclaving in a pressure cooker this is preferred to chemical sterilization of instruments at the last minute With a little thought and preparation a surgical kit or bag can be made to include, in addition to sterile in struments, all the other equipment neces sary for clean surgery on the farm (Brad bury, 1955) In many cases it is feasible to do swine surgery at the office or hospital, in which case there will be more control over the general cleanliness and asepsis in our operations

General anesthesia is preferred and the pig is positioned in dorsal recumbency with the head some 6 to 8 inches below the level of the tail Some operators prefer a field block of local anesthesia, eliminating the danger of overdose of general anes thesia and having the advantage of get ting the pig on its feet immediately fol lowing surgery (Bullard, 1950) Epidural anesthesia is satisfactory for this operation but is not as commonly used as the above two methods (See Chapter 15 for details on anesthesia, restraint, and preparation)

An elliptical skin incision is made around the hernia to include the amount of skin to be removed and any abscess that may be present A high percentage of hernias are involved with abscess forma tion and adhesions In males a U shaped incision can be employed, the ends of the

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U being lateral to the prepuce (Guard, 1948) The incision over the hernia is connected to the ends of the U, allowing the prepuce to be reflected backward to facilitate isolation of the hernial ring With blunt dissection through the subcutaneous and areolar tissue the hernial ring is isolated and cleared in an area of 1 to 1½ inches around the ring

The peritoneal sac is now opened at the neck the contents examined, and the intestines and omentum replaced in the abdominal cavity Adhesions may be present which necessitate freeing the intestines by gentle manipulation with the finger tips Omentum may be adherent to the fundus of the sac and involved in abscess formation in which case it is necessary to ligate and amputate the omentum removing it with the isolated skin Occasionally the loop of intestine may be so involved in the adhesions and abscess that it cannot be freed without rupturing the wall of the intestine, in which case an end to end anastomosis is necessary

The contents of the hernial sac being replaced the peritoneal sac is now completely dissected from the edges of the hernial ring and discarded Others prefer to ligate or stitch through the peritoneal sac at the neck amputate above the line of sutures and reflect the stump back into the peritoneal cavity Using No 2 catgut, the hernial ring is now closed with a series of mattress sutures placed in such a manner that the edges of the ring will overlap (Spivak 1947) The sutures should be placed about ½ inch apart and all sutures put in before they are drawn up snug and tied This method will leave a free edge of the upper flap which should be sutured to the outer surface of the lower flap by a few interrupted sutures The suture line should be checked for gaps and additional small sutures added if necessary The skin incision is closed with suture material of choice We prefer small interrupted mattress sutures of silk and cover the incision line with collodian or plastic spray bandage Antibiotics under the skin along the incision help to control local infection Aftercare consists of keeping the pig in a

clean area with limited exercise and reduced diet until healing is complete

TUSK REMOVAL IN BABY PIGS AND BOARS

The sharp small tusks of baby pigs are clipped to help prevent scratching subsequent infection, and inflammation of the sow's udder and teats, and also to prevent bite infections resulting from the pigs fighting among themselves The practice of clipping teeth of baby pigs varies from herd to herd Some swine raisers do it routinely, some never clip teeth and others only when it appears to be required The tusks on baby pigs are best clipped when the pig is 2-3 days old, although it can be done at any age A pair of side cutting pliers is the instrument of choice and special baby pig teeth nippers of this type are available from farm supply houses A bone cutting forceps is a good instrument to use, or a pair of ordinary electricians pliers works well Care should be taken to avoid cutting the teeth too close to the gum line so that the teeth will not be cracked or broken below the gum A sharp file may be used also to dull the sharp points of baby teeth This method prevents the crushing of the tooth and possible deep infection

The large tusks on mature boars are cut to protect the men handling the boars and to prevent injuries produced through fighting Mature boars are detusked at any age it is deemed advisable These large boars need to be restrained by a method that will hold their heads steady Two ropes or lengths of obstetrical chains can be used around the snout above the tusks and stretched between two posts Other methods of restraint can be used as is convenient (see Chapter 45 on restraint) Placing a block or wedge of wood of proper size as far back across the molars as possible will keep the mouth open A hacksaw is used to cut through the tusks, the cut being made ½ to ¾ inch above the gum line The cut should be made from the inside of the mouth outward to follow the curve of the tusk and to reduce the possibility of injury to the lips and mouth Bolt cut

ters can be used to clip the large tusks care being taken not to crack the tooth below the gum line The tooth should then be smoothed with a file or horse tooth float Other methods such as cracking the tusk off with a blow of a hammer or breaking it with a chisel and hammer are faster but more crude and are not recommended because of the danger of damage to the alveolus and jaw bone Large boars commonly go off feed for a few days following tusk removal but recovery is rapid with no special aftercare

NOSE RINGING

Rings are placed in the noses of swine to help prevent excessive rooting in pastures and lots and under fences The technique is simple and is commonly done by the herdsman The special ring and hog ring pliers are available from supply houses and hardware stores The small pig is held between the knees of an assistant who holds the forelegs with the snout presented to the operator Large hogs can be restrained with a hog holder or any other method of restraint that is convenient (see Chapter 15) The rings are placed in the circular margin of the snout above the nostrils and about ¼ to ½ inch apart using from 1 to 4 rings depending on the size of the pig Rings may be inserted in pigs 8 weeks of age or at any age thereafter but not before the pig is weaned Care should be taken to prevent placing the rings too deep thus causing pressure necrosis The technique of placing the rings in the skin of the septum between the nostrils is not recommended as it does not seem to be as effective a method of preventing rooting Rings which have been worn or torn out can be replaced as required

cava has greatly facilitated the efficiency of swine disease programs and research

Anticoagulants

As indicated by Coffin (1953) anticoagulants are necessary for cell counts hemoglobin and various blood chemistry determinations A 10 per cent solution containing 4 per cent of potassium oxalate plus 6 per cent of ammonium oxalate is most commonly used The ratio of this solution to blood is 0 15 cc per 5 cc of blood The proper amount of solution is placed in tubes for the quantity of blood desired and then evaporated to dryness in a hot air sterilizer or ordinary baking oven set at low temperature Other anticoagulants such as sodium oxalate potassium oxalate and sodium citrate may be used at the rate of 2–4 m$_g$ per cc of blood These also are added to tubes in solution and evaporated to dryness

The anticoagulant of choice depends upon the blood chemistry determination For example blood for nonprotein or urea nitrogen should be collected in potassium or sodium oxalate while sodium fluoride is used at the rate of 6 mg per cc of blood for blood sugar determinations.

to the laboratory because of expense, time, and inconvenience both to the veterinarian and to the owner if one hemolyzed sample is found In their opinion the only consistently satisfactory samples are those obtained by aspiration of clear serum with needle and syringe after the blood clot has contracted and loose blood cells have settled

It is important to remember that natural contraction of a clot is impeded by low temperature Therefore, maintaining the blood sample near body temperature for several hours prior to refrigeration enhances clot contraction

Techniques for Venipuncture

The technique employed varies according to the purpose for which the blood vessel is being entered The three routes employed are ear veins, anterior vena cava, and tail vein

RESTRAINT

The problem of restraint is most vital in securing adequate blood samples with little risk to the animal If the animal is properly restrained, no difficulty should be experienced by a qualified operator in obtaining samples For bleeding or making intravenous injection, many temporary or permanent improvisions can be made using chutes, 'wedge' or 'V' type troughs, holders, or crates for restraining swine (see Chapter 15) In addition to the methods therein discussed three commonly used techniques that are employed at Pennsylvania State University will be described

When the pigs are too big to be held and other means of restraint are inconvenient, a workable procedure for procuring small pipette samples is to crowd a small number of the pigs into a corner where they will commonly huddle Obtain the samples from the ears of those on top, and by subsequent reshuffling of the group all are bled readily with a minimum of effort. A bleeding ear generally is sufficient to identify an animal as having been bled

Bleeding small pigs from the anterior vena cava can best be accomplished by restraining them in a dorsal recumbent position on the ground, floor, table, or over an assistant's knee as shown in Figure 48 1 The prevailing bleeding environment largely determines whether the assistant may stand, sit, or squat when restraining the pig Large pigs are best restrained by snubbing them to a post, as discussed in Chapter 45, and obtaining the sample as shown in Figure 48 2 In obtaining blood daily, a technique routinely used is that of placing the pig in a dorso recumbent position (Fig 48 3), with one assistant on his knees straddling the pig immediately in front of its rear legs (usually only gilts are handled in this manner) With smaller pigs, the assistant may hold both front feet towards him with one hand, and the head down with the other For larger gilts or those more difficult to handle, a second assistant immobilizes the head

TAIL BLEEDING

Tail bleeding is not commonly practiced since methods for bleeding from the anterior vena cava have been perfected Tail bleeding is primarily used in commercial serum plants and essentially involves cleaning of the tail (shaving is best), disinfecting, and excising a distal segment with a sharp instrument

EAR BLEEDING

Ear veins are readily observed in a white eared pig The veins are generally located in three portions of the ear One vein courses along the outer edge, another in the middle, and the other about an inch from the medial margin or top of the ear The latter vein is usually more deeply embedded and consequently is used less than the other two in bleeding or surgical techniques

One method involved in ear bleeding consists of piercing or severing one of the veins and/or the accompanying artery This may be accomplished with a sterile hemolet, Bard Parker No 11 blade, or any other sterile sharp piercing instrument following proper cleansing of the area Blood may be aspirated directly into a pipette and mixed with proper diluent or allowed to flow into a tube Blood samples

FIG 48 1 — Technique for bleeding from the anterior vena cava of a small pig

FIG 48 2 — Method for obtaining a blood sample from the anterior vena cava of a large pig

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FIG 48.1 — Technique for bleeding from the anterior vena cava of a small pig

FIG 48 2 — Method for obtaining a blood sample from the anterior vena cava of a large pig

so obtained are often inadequate in amount for testing and are grossly contaminated. Spontaneous hemolysis tends to occur as the blood flows onto the surface of the ear.

Another method of ear bleeding consists of occluding a vein by an intestinal forceps or by digital pressure. The skin is then stretched across the vein to immobilize it, and the needle with the bevel up is inserted A sharp 16- to 20 gauge needle may be used, depending upon size of the animal If the needle has been properly inserted and held, the pressure may be released to allow recirculation prior to obtaining a blood sample. Gentle traction is applied to the syringe plunger until the desired amount of blood is obtained. The needle is then withdrawn, removed from the syringe, and the blood discharged into the proper container. Blood transfusions, hyperimmunization, or any therapy or surgical technique requiring the injection of 100 cc. or more can be made by using the ear vein, 100-cc. syringes, and 16 gauge needles.

BLEEDING FROM ANTERIOR VENA CAVA

The anterior vena cava method of bleeding is the most common among veterinarians. The method as first described by Carle and Dewhirst (1942) is safe, rapid, and easy to perform.

The anterior vena cava lies in the thoracic inlet between the first pair of ribs and gives rise to external jugulars and the right and left brachial veins. If the pig is in a dorso-recumbent position the needle is inserted on the right side ½ to 2 inches (varies with size of animal) from the apex of the cartilage to the base of the ear. The point of the needle is guided inward, downward, and backward to the entrance of the thorax between the first pair of ribs.

According to Hoerlein *et al.* (1951), possible injury to the phrenic nerve is of great importance. On the left side it courses for a short distance parallel to, and in close proximity with, the external jugular vein, thereby making it vulnerable to injury by the bleeding needle. The right phrenic nerve has the same origin as the left but is partially protected for some distance by the scalenus ventralis muscle. As the nerve enters the thoracic inlet, the right brachial vessels lie superficial to the nerve, giving the nerve on the right side greater protection.

A glass syringe and a needle of proper length and gauge should be used. The choice of length and gauge of the needle depends primarily on the animal's size.

FIG. 48.3 — The method employed for obtaining larger quantities of blood from the anterior vena cava of a gilt.

Blood has been obtained routinely from pigs weighing about 40 lb to more than 200 lb with a 3 to 4 inch, 16 gauge needle Needles of 2 to 3 inches and 17 to 19 gauge are used on all pigs under 40 lb , with the 19 gauge being used on the smallest pigs A 6 inch, 12 gauge needle has been used on adult hogs and occasionally on pigs as light as 100 lb with no ill effects this gauge and length is, however, not recommended for routine use on the farm Hoerlein *et al* (1951) successfully used 1½ inch, 20 gauge needles for pigs from birth to 50 lb and 3½ to 4½ inch, 17 to 19 gauge needles on all others The lighter gauge needles however, are too limber and often kink becoming useless after a few difficult bleed ings The stiffer 16 and 17 gauge needles have proved more durable and accurate in penetrating to the point desired

Bleeding from the anterior vena cava as with any surgical procedure, is not de void of danger to the animal Since pigs lack sweat glands, they are very suscep tible to heat stroke, especially in hot and humid weather Extremely nervous animals should be caught, restrained and bled as quickly as possible even if the temperature is below 70° F If severe dysp nea should occur immediately upon re lease the pig should be kept as quiet as possible This distress is often temporary and usually disappears completely in 12 hours

Occasionally animals with hemophilia may continue to bleed into the tissues of the thoracic inlet, neck muscles and sub cutaneously at the point of penetration until death results from asphyxiation

Individual pigs have been bled contin uously at the rate of 1–3 cc of blood per pound of body weight weekly and often twice weekly During this period they have remained healthy and have grown at ap parently normal rates from 40 lb to more than 200 lb None of these pigs has died and for this reason, the degree of connec tive tissue infiltration and damage to the anterior vena cava has not been deter mined

A modified version of Sippel s (1949)

procedure for bleeding a swine herd is as follows

1 Restrain the animals by method of choice
2 Cleanse the neck with disinfectant solution and cotton
3 Have several needles of proper gauge and length
4 Have several glass syringes (prefer ably 10 cc)
5 Withdraw the blood sample
6 Remove the needle from the syringe (to prevent hemolysis)
7 Slowly discharge the blood down the side of the container
8 Rinse the needle and syringe in cold water
9 Place the needle in a pan of antiseptic solution
10 Attach a second needle to the syringe and then rinse in sterile sodium citrate
11 Make the necessary records
12 Proceed to next pig

ABSCESSES

Abscesses in swine are not a rarity and most veterinarians having a large animal practice could write several interesting case histories relative to this problem Of pri mary interest under this heading are those abscesses which can be treated surgically

Occurrence

Of our domestic animals, swine probably suffer the most from poor husbandry and management This consequently predis poses an individual animal or herd to ab scess problems which may arise from the pyogenic infection of bruises or pene trating wounds

Mastitis bruises and various kinds of wounds make the sow s udder a common site for abscess formation The sharp teeth of suckling pigs are responsible for the majority of the wounds

Following parturition hematomas and lacerations of the vulva of gilts and sows commonly predisposes them to abscess conditions, especially when the farrowing environment is undesirable

Injuries incurred from swallowing mate rials like wire, tin nails and glass cause abscessation in the pharyngeal area In ad dition, pharyngeal abscesses are caused by the careless use of a balling gun in ad ministering medicine or by an oil can spout which farmers often use for giving baby pigs medication for anemia The diverti culum pharyngeum is the anatomical fea ture that greatly enhances abscessation in the pharynx by allowing materials to be come lodged

High feeders and drinking fountains with sharp edges frequently cause injuries which result in jowl abscesses Overcrowd ing encourages fighting and tends to make the problem more acute by increasing the number of cuts, abrasions, and lacerations which can become infected Pigs often rub themselves on anything that is convenient to use, especially fence posts and door frames It is important to keep these ob jects free of sharp edges or protruding nails Puncture wounds caused by pitch forks and other sharp equipment also may cause abscesses

Pharyngeal region abscesses recently have been investigated more thoroughly and merit consideration as a specific disease en tity Collier (1956) reported the group E streptococci are regularly associated with abscesses that occur in this region Other workers Newsom (1937), Stafseth and Clinton (1911), Snoeyenbos et al (1952), and Smith (1956), have also reported on this condition

The reader is referred to Chapter 25 for a discussion on abscesses in the pharyngeal area in association with *Corynebacterium equi* infections

In young pigs umbilical abscesses are a constant problem Umbilical hernias also are often complicated by abscessation

Etiology

The microorganisms commonly associ ated with suppurative processes, as listed by Runnells (1954) are the various staphylo cocci and streptococci, *Escherichia coli*, *Shigella equiruli*, *Corynebacterium pyo genes C pseudotuberculosis Pseudomonas*

aeruginosa, *Spherophorus necrophorus*, and *Actinobacillus lignieresi* Collier (1956) in his survey of 492 exudates from the pharyngeal region found that 85 6 per cent of the exudates contained a beta hemolytic *Streptococcus* belonging to Lancefield's serologic group E The other organisms isolated and their percentages are as follows *Corynebacterium pyogenes* (12 8), *Pasteurella multocida* (5 89), *Pro teus ammonia* (3 25), *Streptococcus equis imilis* (1 42), *Escherichia coli* (1 02), *Streptococcus zooepidemicus* (0 81), *Ac tinomyces bovis* (0 41), *Spherophorus necrophorus* (0 41), *Proteus vulgaris* (0 20), *Salmonella typhimurium* (0 20), *Staphylococcus aureus* (0 20) Hollister (1956) isolated *Streptococcus agalactiae* from a cervical abscess

Various inorganic and organic chemical substances also can cause suppuration A few such substances are mercuric chloride, zinc chloride, turpentine, and croton oil Many others such as caustics, acids, alka lies, and insecticide sprays, under varying conditions of usage or by accident and aided by microorganisms, can cause sup purative conditions

Pathology

Purulent exudate (pus) that becomes a sharply circumscribed focus in the tissue is an abscess Abscesses are generally clas sified as hot or cold," being acute or chronic processes, respectively A fu runcle (boil) is a cutaneous abscess of a few millimeters or less in size A car buncle resembles a boil but is larger and has a flat surface and multiple openings A pustule' is a very minute abscess in the malpighian layer of the skin

Characteristic pus is thick, creamy, smooth, and varies in color from yellow to yellowish green It should be odorless and contain no threads of necrotic tissue The pus of swine has a tendency to be caseated or fluid as compared with that of avian origin The latter usually is cheesy because it contains an antitryptic enzyme Run nells (1954) states that an enzyme pres ent in lymph inhibits the proteases of

polymorphonuclear leukocytes therefore no suppuration occurs when serum is plentiful

According to Collier (1936) the pharyngeal abscesses caused by the beta hemolytic streptococci belonging to group E have a characteristic exudate that is nonodorous is distinctly greenish in color and varies in consistency from creamy to glutinous The size varies from less than 1 cm to more than 10 cm in diameter, the smaller ones being embedded in lymph nodes of the region

Clinical Signs

The symptoms of abscessation depend primarily on the size and location of the abscess and may vary from none to prostration and death An animal may show a few or all of the cardinal signs of inflammation It may be lame or merely show reluctance to move about Difficulty in defecation and micturition may be observed if the abscesses are located on the vulva or in or about the perineum Soreness of the pharynx and interference in swallowing causes anorexia Pressure exerted by an abscess in the throat area may result in dyspnea coughing and discharge from the nose Due to soreness of the udder a sow may refuse to nurse her pigs

Prevention and Control

Needless to say, good husbandry and management practices greatly reduce the incidence of abscesses One of the more important preventive measures is the removal of sharp or penetrating objects in the pens or houses Nails broken wire fences and sharp edges on sills, drinking fountains and feeders are the more apparent offending objects A clean well bedded farrowing house and the removal of the tusks of baby pigs greatly reduces injuries to a sow's udder

Feed should be handled with care to avoid incorporation of foreign objects that may lodge in or penetrate the pharyngeal area Strict sanitary measures concerned with feeding are very important as is exemplified by the report of Hollister (1956)

who isolated *Streptococcus agalactiae* from cervical abscesses The circumstantial evidence of the source of infection implicated whole milk which had been rejected on deck inspection in a local dairy The containers used to transport the milk were used repeatedly without cleaning

Isolation of infected animals is a very important practice This is often neglected because of the increased labor and inadequate housing facilities Animals with abscesses that have ruptured or have been surgically opened should be isolated Smith (1936) quoted R A Packer on the isolation of hemolytic streptococci from cervical abscesses The condition was reproduced by adding the organisms in the feed and water but not by intravenous inoculation

Depopulation and disinfection of premises may be necessary in order to remove certain etiological agents as a herd problem on a particular farm Collier (1954) reported that depopulation disinfection of premises and introduction of supposedly abscess free breeding stock failed to eradicate the beta hemolytic streptococci of Lancefield's group E from one farm

Treatment

An abscess should not be surgically incised until it is ripe It is considered to be ripe as soon as it softens in the center and the bristles (if present) fall out Application of liniment or poultices hastens the suppuration of an abscess and thus enhances the ripening process Either an antiphlogistine or flaxseed with an added antiseptic is a useful poultice

Hematomas are often mistaken for abscesses consequently the cavity of the abscess in question should always be explored with a hypodermic needle and syringe

Treatment generally falls into three main categories (1) providing drainage (2) controlling microorganisms and (3) stimulating body defenses When the abscess is ready to be opened it should be incised in a manner that will give the best drainage This is followed with irrigation

to wash the cavity of all pus Antiseptic solutions or physiological saline with antibiotics generally are used for this purpose One should avoid damage to granulation tissue that is forming Dakan's solution is used most often when necrotic tissue needs to be removed by daily irrigation Soak gauze in a tincture of iodine and place it in the abscess for several minutes up to 24 hours if good drainage cannot be obtained The procedure to be followed will depend upon the size of the abscess and its pyogenic membrane The membrane will

be destroyed and slough, and healing will proceed much faster Cotton may be substituted for gauze, but gauze is preferred Dry absorbent powders like boric acid, charcoal, and lime mixtures are sometimes put in packs

In controlling microorganisms, one must keep the animal as free as possible from feces, mud, or dirt Antiseptics and antibiotics also may be employed

Internal medication with biologics or antibiotics serves to stimulate the appetite and body defenses

REFERENCES

Umbilical Hernia

BRADBURY, R. H 1955 Surgical kits for ready field use by practitioners Proc. 92nd Meet Amer Vet Med Assn p 581
BULLARD J F 1950 A comparison of surgical procedures in reducing umbilical hernias in swine Jour Amer Vet Med. Assn 116 101
FRANK, E. R. 1942 Veterinary Surgery Notes Burgess Publ Co Minneapolis
GUARD, W F 1948 Surgical Techniques for Veterinary Students Edwards Bros. Inc., Ann Arbor, Mich
SRIVAR J L. 1947 The Surgical Technic of Abdominal Operations 4th ed Charles C Thomas Springfield Ill

Abscesses

COLLIER J R 1951 A survey of beta hemolytic streptococci from swine Proc Amer Vet Med Assn 88 169
——— 1951 Swine jowl abscesses Iowa Vet. 25 16
——— 1956 Abscesses of the pharyngeal region of swine Amer Jour Vet Res 65 610
HOLLISTER, C. J 1956 Streptococcus agalactiae isolated from cervical abscesses in swine Jour Amer Vet Med Assn 128 555
NEWSOM, I E. 1937 Strangles in hogs Vet. Med. 32 137
RUNNELLS R A 1954 Animal Pathology 5th ed The Iowa State University Press Ames Iowa
SMITH H C. 1956 Cervical lymph gland abscesses Vet Med 51 402
SNOEYENBOS G H., BACHMAN B A., AND WILSON E. J 1952. Abscesses associated with group F Streptococci Jour Amer Vet Med Assn 120 151
STALHEIM, H J., AND CLINTON I 1941 Lancefield group E Streptococci in cervical abscesses of swine Jour Amer Vet Med Assn 77 468

Bleeding Procedure

CARLE, B N., AND DEWHIRST, W H., JR. 1942. A method for bleeding swine Jour Amer Vet Med. Assn 101 195
COFFIN DAVID L. 1953 Manual of Veterinary Clinical Pathology. 3rd ed Comstock Publishing Associates, Ithaca, N Y. p 119
HOFFLIN A B., HUBBARD E D., AND GETTY, R. 1951 The procurement and handling of swine blood samples on the farm. Jour Amer Vet Med. Assn 119 557
SIPPEL, W M 1949 Bleeding hogs from the vena cava Jen Sal Jour 32 (Jan Feb) 4

Nutrition, Feeds, and Management

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CHAPTER 49

Nutritional Deficiencies

Swine require proper nutrition throughout
their lifetime, during health as well as dis
ease Growing pigs in particular require
specific nutrients for optimum growth and
feed efficiency These nutrients are es
pecially necessary during the post weaning
period, when pigs are changed abruptly
from a diet composed largely of milk, or
supplemented with milk, to a diet that may
lack certain essential nutrients Sows, like
wise, have critical nutritive requirements
that must be provided especially during
gestation and lactation to insure the pro
duction of large healthy litters

Moreover, since pigs feed close to the
ground and are in close contact with ex
creta and with other swine, they are ex
posed to the hazards of more infections
and diseases than any other livestock For
this reason, in addition to being supplied
the nutrients necessary for normal physio
logical activities during growth, repro
duction, and lactation, they must also be
given the nutrients they require during
diseases, stresses, toxicities, and nutrient
imbalances Feeding swine under practical
farm conditions is therefore a broader and
more complicated problem than is gen
erally realized, because poor nutrition can
result from causes other than deficient ra
tions as such

This chapter discusses only some of the
important nutritional problems encoun
tered especially under disease and actual
field conditions For more detailed dis

cussion of swine nutrition, the reader is
referred to the excellent texts of Morrison
(1956) and Maynard and Loosli (1956)

NUTRITION AND INFECTION

It is often stated that well nourished
swine resist the various infections better
than do poorly nourished swine This be
lief is not supported by experimentation,
but it has gained acceptance probably be
cause swine raisers who use carefully for
mulated and well balanced rations are also
likely to employ sound disease preventive
measures and thus encounter the minimum
amount of infection However, some re
search has demonstrated that pigs fed on
diets deficient in certain amino acids or
protein were less susceptible to hog cholera
than were pigs fed on complete rations
(Whitehair, 1953) Similar results have
been obtained for other viruses in other
species (Jones *et al*, 1946) Thus, while
good rations may decrease susceptibility
to bacterial, parasitic, and other specific
infections they may also leave the animals
more susceptible to viral infections These
possibilities illustrate that before the role
of nutrition in preventing infection can
be stated, the exact cause must be de
termined They also illustrate the complex
relationships that exist between infection
and nutrition.

In general, nutritional deficiencies may
be considered to result from (1) infec
tions that interfere with ingestion, absorp-

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tion or utilization of essential nutrients, and from (2) causes that increase nutritional requirements or lead to the destruction or abnormal excretion of nutrients. This type of nutritional deficiency might be referred to as secondary nutritional deficiency, its nature, nutrients concerned, and treatment will depend primarily upon the pathological processes involved

Nutrition has definite implications in certain diseases of swine, among which are digestive disturbances that apparently are common in many herds. These disorders which perhaps stem from crowded conditions, are an especially important problem in many large herds, where they some times become endemic (Whitehair et al, 1948a) The importance of nutrition in treating such enteric disturbances in swine under field conditions has long been recognized by practicing veterinarians and re ported by Hofferd (1936), Bryant (1938), Wilson (1940), Truax (1941), Steenerson (1942), and Kernkamp (1945) Its importance has also been confirmed under experimental conditions by Whitehair et al (1948b), and Luecke et al (1949) Similarly, Iradkin (1953) has emphasized the importance of nutrition in diarrheal diseases of humans by pointing out that diarrhea is probably responsible for more nutritional deficiencies than any other symptom or group of symptoms In like manner, starvation diets or inadequate diets cause more therapeutic failures in the management of diarrheal diseases than any unwise choice of drug or combination of drugs

Cause and Treatment

Often the specific and dramatic response to nutritional therapy leads one to conclude that digestive disturbances are due primarily to inadequate rations However, there is very little evidence that deficient rations are the primary cause of these disturbances For example, feeding a wide variety of nutrients and supplements in large amounts did not prevent a characteristic digestive disturbance (Whitehair,

1951), nor was it produced by feeding rations deficient in amino acids and vitamins and containing antibiotics to suppress possible infection (Hillier and Whitehair, 1952) On the other hand, the role of infectious agents has not been extensively or adequately investigated While several infectious agents have been implicated in these disturbances, others undoubtedly will be incriminated when additional research efforts are applied to this problem Deficiencies are often cited as causes simply because the investigator is unable to isolate a certain pathogen or to reproduce the disease with filtrates Frequently the animal may have become infected much earlier, even during the early nursing period, hence the primary pathogen can no longer be isolated Likewise, in conducting transmission experiments one should make certain that the pigs used have not been exposed previously and are immune or that they are of disease free origin

Diet therapy will depend, of course, on the duration and pathogenicity of the enteric infection It will consist primarily of restoring the appetite and replacing the nutrients lost Table 49 1 shows how an experimentally produced specific infection decreased food consumption and increased fecal nutrient losses in young pigs It might be added that while the nutrient losses in this experiment were quite marked, the clinical symptoms of these pigs when exposed to an enteric infection were

TABLE 49 1

EFFECT OF TRANSMISSIBLE GASTROENTERITIS INFECTION IN PIGS ON FOOD CONSUMPTION AND EXCRETION OF NUTRIENTS*

Items	Controls	Infected
Number of pigs	6	6
Daily food consumption (gm)	160	94
Daily fecal excretion		
Water (gm)	1 3	40 7
Nitrogen (gm)	0 03	0 56
Sodium (mg)	1 1	49 8
Potassium (mg)	5 0	231 0

* Revised table from Reber and Whitehair (1955)

not nearly as serious as has been observed under practical conditions

Other infections can also be presumed to interfere with nutritive requirements of pigs Besides affecting the metabolism of specific nutrients they may depress appetite This action starts a vicious cycle of insufficient food consumption and consequently a complex nutritional deficiency In the final analysis the treatment of digestive or other diseases must include the nutrients necessary for restoring the animals to normal growth as quickly as possible as well as treatment to suppress or eliminate the specific infectious agent

NUTRITIONAL DEFICIENCIES IN GENERAL

Nutritional inadequacies may vary from mild deficiencies without obvious symptoms other than impaired growth and feed utilization to marked deficiencies with a definite clinical syndrome and lesions Unfortunately the symptoms lesions and biochemical changes observed under *experimental* conditions are sometimes difficult to apply to field problems because specially devised rations are employed to produce a more absolute and acute deficiency Under *field* conditions an incomplete deficiency exists with a more chronic course complicated with other deficiencies and factors Certainly some nutritional deficiencies produced in swine under experimental conditions are not likely to occur under field conditions either because the necessary dietary requirements are so small that deficiencies would be difficult to produce or because the commonly used feeds would supply adequate amounts of the nutrient On the other hand it would be folly to predict the practical applications of basic research Zinc for instance considered to be unimportant in swine nutrition by research workers (for a long time and with good reason) is now recognized as an essential nutrient in the control of parakeratosis

Nutritional deficiencies encountered under field conditions usually exist as complex deficiencies Their general symptoms are poor growth rate reduced appetite unthriftiness lameness and disturbances of the haircoat and skin These symptoms especially the first two are so general that in many herds they may not be detected at all If accurate records could be obtained these symptoms would be helpful in detecting the mild deficiencies undoubtedly common in many herds Complex deficiencies can be detected only by carefully reviewing the diet as well as the clinical symptoms pathological findings and perhaps biochemical determinations Table 49 2 illustrates the total nutrients and the amount supplied by each feed in a simple ration for swine in comparison to the daily requirements Note the marked deficiency of calcium and borderline amounts of phosphorus riboflavin and pantothenic acid It is also evident that while alfalfa meal makes up only a small amount of the total ration the contribution of vitamins and calcium is considerable Management and physical equipment also play a part Adequate well-designed feeders palatable rations and sufficient exercise all help ensure good nutritional performance

WATER

Water is important but often neglected in swine feeding Swine can live for many days without food but only a few days—perhaps in some environments only hours—without water Water is an important structural component of cells and tissues and plays a major role in cellular metabolism by carrying dissolved or emulsified nutrients to the cells and carrying secretions and excreta away (For a review on water metabolism and requirements of farm animals see Leitch and Thomson 1944)

Water is also important for regulating body temperature Heavy pigs are especially susceptible to heat prostration because they have a layer of fat which retards the escape of heat and because they lack ordinary sweat glands Consequently heavy losses of water accompanied by electrolytes in disturbances such as gastroenteritis produce serious physiological and

pathological consequences, including dehydration, rapid weight loss, and anorexia (Marriott, 1950).

CARBOHYDRATES

The group of foodstuffs called carbohydrates supply the major energy requirements for the many body activities. For this reason, carbohydrates compose 70 to 80 per cent of swine rations by weight. Starch, the principal carbohydrate in swine rations, is broken down by the digestive enzymes into the end product, glucose, a simple sugar After absorption, glucose is utilized immediately for energy or transformed to and stored as fat. Another simple sugar in milk, lactose, has special nutritive properties in that it promotes an acid or favorable type of fermentation in the intestine, is more slowly absorbed than the other simple sugars, and enhances the absorption of calcium and phosphorus. For these reasons, and the fact that baby pigs do not utilize dietary sucrose (Becker *et al*, 1954b), lactose is the carbohydrate of choice in compounding synthetic rations for baby pigs.

Crude fiber or complex carbohydrates from hay or other roughages are utilized only to a limited extent by growing pigs. Legume hays are good sources of many vitamins and minerals and are used extensively in practical growing rations at levels of 5 to 10 per cent (depending on quality) of the total ration. For mature stock, especially brood sows, the amount fed may be increased 15 to as much as 50 per cent. The higher levels would be indicated for sows self-fed during gestation to prevent them from becoming excessively fat.

Besides supplying energy, the carbohydrates supply special protective and detoxifying powers to the liver (Soskin and Levine, 1952). When liver glycogen stores are low, animals are much more susceptible to various poisons such as carbon tetrachloride, chloroform, or arsenic, and to some of the toxemias of microbial origin. The mechanism of this detoxification is not clearly understood, but part of it is presumed to consist of a conjugation of the toxic substance with carbohydrate com-

TABLE 49 2

VITAMIN,* MINERAL, AND PROTEIN CONTENT OF 3 MAJOR COMPONENTS OF A SIMPLE SWINE RATION (3 2 LB PER DAY) IN COMPARISON TO DAILY REQUIREMENTS†

Component of Ration	Amount of Component in 100 lb of Ration	Protein Supplied by Component	Tryptophan Supplied by Component	Lysine Supplied by Component	Methionine Supplied by Component	Ca Supplied by Component	P Supplied by Component	Thiamine in Daily Ration	Riboflavin in Daily Ration	Niacin in Daily Ration	Pantothenic Acid in Daily Ration	Carotene in Daily Ration
	(lb)	*(lb)*	*(lb)*	*(lb)*	*(lb)*	*(lb)*	*(lb)*	*(mg)*	*(mg.)*	*(mg)*	*(mg)*	*(mg)*
Corn	72	6 19	05	15	16	01	19	4 1	1 1	23 5	6 2	4 6
Soybean meal (43%) .	20	9 20	17	60	10	.05	14	3 9	1.1	7 3	6 5	6
Alfalfa meal, sun-cured	5	88	01	04	02	07	01	2	8	2 6	2 0	3 5
Total	97‡	16 27%	23%	79%	28%	13%	34%	8 2	3 0	33 4	14 7	8 7
NRC (1953) † . .		16 0%	.20%	1 00%	60%	.65%	.45%	1.6	3 2	19.2	16.0	1 0

* Vitamin amounts and requirements are given as mg. per 3 2 lb. of ration.
† Requirements of NRC = Swine requirements, National Research Council, 3 2 lb. feed daily for 50-lb. pig.
‡ Plus 3% for added minerals, vitamins, animal protein, etc.

pounds, transforming the toxic factor into a relatively innocuous substance

While a wide variety of feeds supply carbohydrates satisfactorily for swine feeding the usually available and economical sources are corn and the small grains Formulating an adequate swine ration is primarily a matter of including supplements to provide the amino acids, minerals and vitamins lacking in the grain portion of the diet.

PROTEINS

Nutritionally, proteins are usually considered the most important of the three classes of organic nutrients Protein is the main component of the soft tissues and organs of the body, it is a structural constituent of the cells making up these parts and is vitally important in many active biochemical substances, such as hormones enzymes, immune bodies, and hemoglobin Proteins are also of considerable importance in the resistance to and recovery from various diseases In a deficiency, for example, the capacity to fabricate antibody protein is low, the production of leukocytes and lymphocytes is decreased and the bone marrow and lymphoid tissues are depleted (Miner, 1955, Cannon, 1918, 1950)

In nutrition, the individual amino acids that make up the simple proteins are of most importance Of the more than 20 amino acids that have been isolated from proteins, 10 are essential for swine (National Research Council Report, 1953) Of these 10, tryptophan lysine, and methionine are particularly important These amino acids may be present in insufficient amounts in the feeds commonly used in swine rations Recent studies (Becker et al, 1954, Meade, 1956a) using typical diets have suggested levels of approximately 0 13, 0 66 and 0 25 per cent respectively for tryptophan lysine, and methionine as adequate for growth and acceptable nitrogen retention in pigs These requirements are markedly lower than those suggested earlier (Table 49 2) and probably represent a closer figure to the actual requirement Proteins vary in

their amino acid content and those containing amino acids in amounts that parallel the body requirements are referred to as proteins of high biological value

After proteins have been digested and the individual amino acids absorbed protein metabolism is considered to be in a state of dynamic equilibrium between the plasma proteins of the blood and the cellular protoplasm of the various tissues organs and hemoglobin Thus the proteins in various tissues are continually synthesized and broken down Likewise there is a continual loss of protein from the body by deamination in the liver and excretion of the nitrogenous products in the urine Synthesis of body proteins involves many factors such as energy minerals and vitamins The amino acids required to fabricate a specific protein must be present not only in the right amounts but also at the right time (Lggert et al 1953) Thus the theory (Cannon 1918) applies that a deficiency or absence of one amino acid limits protein synthesis

Infections seriously disturb protein metabolism This is a secondary protein deficiency, and its pathological consequences and symptoms are much more severe than a primary deficiency A secondary deficiency is brought about by (1) anorexia (2) partial or total reduction in food intake (3) loss of protein in secretions various fluids and hemorrhage, (4) an excessive breakdown of tissue proteins and (5) failure of the body to synthesize proteins The symptoms and tissue changes depend primarily on the pathogenicity and duration of the infection and the tissues involved The expected pathological effects are anemia hypoproteinemia leukopenia rapid weight loss slow wound healing increased susceptibility to certain infections and tissue atrophy

Proteins are not stored like other nutrients However there are certain deposits or reserves in the protoplasm of certain tissues such as muscle and the tissues of the liver, that may be used during periods of inadequate protein intake Thus protein deficiency is first reflected in the

plasma protein values and later in lowered hemoglobin levels. Symptoms are anemia, atrophy of tissues, and weight loss.

Using purified type rations under experimental conditions, most of the essential amino acids required by swine have been identified or determined. Under field conditions, however, no symptoms are likely to be observed except the general ones of impaired growth and unthriftiness. Methionine deficiency will reduce feed efficiency (Whitehair and MacVicar, 1952). Growing pigs, unlike ruminants, do not utilize urea as a source of nitrogen (Hanson and Ferrin, 1955).

FATS

In swine nutrition, fats are not considered to have any special importance other than a place in general nutrition This includes: (1) their importance in the metabolism of the fat-soluble vitamins; (2) a concentrated source of energy; (3) adding palatability to rations; and (4) furnishing the essential fatty acids, linoleic, linolenic, and arachidonic. Witz and Beeson (1951) have described a fatty acid deficiency in swine which was experimentally produced by feeding a diet composed of 0 06 per cent fat. However, practical rations probably would never be this low.

MINERALS

The mineral elements have many vital functions in the body. They are essential components of the skeletal structure and, in combination with fats, proteins, and carbohydrates, make up many important organic compounds. Many of the enzymes have specific inorganic ions present. As soluble salts they have a wide variety of functions such as osmotic pressure, acid base relationships, and characteristic effects on the irritability of muscles and nerves

Calcium and Phosphorus

Calcium and phosphorus are usually considered together since approximately 99 per cent of the calcium and 80 per cent of the phosphorus are found in the bones and teeth.

Rickets in fast-growing young pigs and osteomalacia in sows that are lactating heavily are probably the most frequently observed nutritional deficiencies in swine (Kernkamp, 1925, 1941; Bohstedt, 1926; Mitchell, 1929; Loeffel *et al.*, 1931; Theiler *et al.*, 1937). In pigs, rickets is characterized by various forms of stiffness, unthrifty appearance, poor growth rate, and low calcium and phosphorus blood values. Paralysis, especially of the rear quarters, is frequently observed. The joints show enlargement and are painful. The weight of the body and tension of the muscles cause the long bones to twist or bend various ways or even fracture.

Osteomalacia is usually observed in sows during the middle to the latter part of lactation. The symptoms are various forms of posterior paralysis, lameness, and stiffness (Fig. 49.1). They are usually the result of spontaneous fractures of the pelvic bones, the femur, or the vertebrae in the lumbosacral region. These bones are in various stages of decalcification to meet the demands for milk production and are unable to withstand any sudden contractions of the powerful back muscles, such as might result from slipping or from exertion.

In normal calcium and phosphorus metabolism three factors are necessary: (1)

FIG. 49.1 — Posterior paralysis (or "downer") in sow during fifth week of lactation. Blood calcium values were low, and on necropsy fractures of both femurs and other lesions of osteomalacia were found.

sufficient supply of each mineral (2) suitable ratio between them and (3) vitamin D Of these three factors calcium is the most important one to consider Swine require it in rather specific amounts during rapid growth and heavy milk production It also is most likely to be deficient in swine rations because the concentrates usually used are low in calcium Corn is particularly low in calcium (02%) The recommended calcium phosphorus ratio is usually between 1 1 and 2 1 (Bohstedt 1939) Vitamin D is required in the deposition of the calcium phosphate in the bony matrix In rickets or osteomalacia the proteinic matrix of the bone tissue appears normal but the deposition of calcium phosphate is impaired and osteoid zones are developed around the bone trabeculae Decalcification while evident in both rickets and osteomalacia is usually more extensive in osteomalacia These disturbances cause a wide variety of skeletal defects

At necropsy of rachitic pigs the bones especially the ribs are soft and at times so spongy they may be cut readily with a knife The epiphysis of the long bones is enlarged and irregularly club shaped while the shaft is also irregularly thickened The yellow marrow is red and gelatinous The articular surfaces may be ulcerated and the bones of the vertebral column and pelvis may be rarefied or fractured

In sows a calcium deficiency analogous to milk fever in cows apparently does not occur (Hastings 1955)

Magnesium

Magnesium is also concerned in calcium and phosphorus metabolism and is a component of many enzyme systems High levels of magnesium cause generalized anesthesia and complete muscular relaxation Low levels cause dilation of capillaries extreme nervousness and convulsions Most swine rations contain enough magnesium for requirements Magnesium has an antagonistic action towards calcium An excess in the ration may cause an excessive loss of calcium This apparently is not as much of a problem in swine as in other species Unless

the excess of magnesium is very large usually no harm results

Sodium Potassium, and Chlorine

Sodium and potassium exist in ionic form in tissue and make up the basic part of several enzyme systems which maintain the physiological pH of blood various body fluids and digestive juices Potassium is the main cation of the fluid within tissue cells while sodium is in the extracellular fluid Chlorine combines with hydrogen to form hydrochloric acid which gives the gastric juice proper acidity

Because of its presence within cells a great many physiological roles have been ascribed to potassium Among the important functions is its relationship to the metabolism of muscles and nerves In experimental animals a potassium deficiency is characterized by paralysis and irritability of skeletal muscles While swine require potassium (Hughes 1942) common rations usually contain ample amounts No nutritional problem is presented unless there are unusual losses as in diseases such as gastroenteritis

Sodium and chlorine are associated as common salt in ingestion excretion and many bodily functions such as water regulation osmotic pressure and control of plasma volume Salt is so important for swine that Iowa workers (Evvard et al 1925) emphasized its importance by referring to it as the white gold of the swine kingdom They noted that the lack of salt caused slow growth very poor feed efficiency and a depraved appetite in pigs

The kidneys are an efficient regulatory mechanism which maintains a definite concentration of salt in the blood and extracellular tissues over a wide range of intake Losses are minimized when intake is low excessive amounts are excreted when intake is high

Salt poisoning of pigs may occur if an unusual amount of salt is consumed without fresh water The usual recommendation for salt in swine rations is 0 5 per cent of the total ration

Infections especially those involving the

intestinal tract, cause excessive losses of salt and these losses must be replaced In many illnesses salt helps to restore appetite

Iron and Copper

Iron is an essential component of the he moglobin molecule and several enzymes As a constituent of hemoglobin it has the important physiological functions of oxy gen transport and cellular respiration Cop per is required in the metabolism of iron and in the synthesis of hemoglobin The importance of iron and copper in the treatment and prevention of anemia in young pigs was shown through research conducted at several experiment stations during the 1920 s and 1930 (McGowan and Crichton 1923 Doyle et al, 1927, Hart et al, 1929, Hamilton et al, 1930)

Pigs are born with only a limited store of iron and copper Milk is low in these elements, and unless pigs have access to outside sources, anemia develops in 2 to 3 weeks The hemoglobin levels decrease in the baby pig from values at birth of 8 to 12 gm per 100 ml blood to values as low as 2 to 3 gm in 3 to 4 weeks (See Chapter 2 on hematology for other hemoglobin values) The anemia that results is a hypo chromic microcytic type Anemic pigs show symptoms of poor growth, listlessness, rough haircoat, wrinkled skin, drooping ears and tail, and a paleness of the mucous membranes Fat, well nourished pigs may die suddenly A rather characteristic symp tom is labored breathing or a spasmodic jerking of the diaphragm muscles from which the term 'thumps' arises Necropsy findings are enlarged and fatty liver, thin watery blood ascites, marked dilation of the heart, and enlarged firm spleen Eryth roblastic cells appear in clumps in the bone marrow and liver

Anemia occurs mainly when pigs are kept indoors on concrete floors during the late fall or early spring Feeding iron and copper salts to sows during gestation is not effective in increasing reserves in newborn pigs. Usually the hemoglobin decreases moderately during the first 3 or 4 days of age regardless of whether or not the pigs have access to iron and copper salts With access to iron and copper, however, by 2 or 3 weeks of age the values are equal to or above those noted at birth

The metabolism of iron is rather un usual in that the body has a remarkable ability to conserve iron after it has been absorbed and has become part of the tissues The body retains the iron lost from the destroyed red blood cells and uses it in resynthesizing hemoglobin Absorption is controlled in some manner by the intesti nal mucosa, which is able to accept iron in times of need and reject it when stores are adequate Thus, iron in rations in excess of normal requirements serves no useful purpose and may actually form a complex insoluble salt with calcium and cause rickets However, stress conditions, hemor rhage, and infections increase iron require ments

Anemia in pigs can be prevented by a variety of methods placing clean sod in the corner of the farrowing pen, applying iron and copper salts to the sows udder, or ad ministering iron copper tablets Also avail able commercially are injectable iron com pounds which are very effective

Pigs apparently are not susceptible to the toxic factor in trichloroethylene extracted soybean meal that produces aplastic anemia in cattle (Hanson et al, 1956)

Iodine

Iodine is required for the proper devel opment and functioning of the thyroid gland and is an indispensable component of the hormone, thyroxin, which controls the rate of energy metabolism In iodine defi ciency there is a hypertrophy of the follicu lar epithelium in an effort to produce more thyroid hormone and a corresponding en largement of the thyroid gland (simple goiter)

It has been known for some time (Welch, 1928) that swine in the goiter area of the Great Lakes and the Pacific Northwest were susceptible to an iodine deficiency Re ports (Andrews et al, 1948, Slatter, 1955) indicate that a deficiency may also occur in central Indiana Andrews and associates

logical factors may be involved in this disturbance Zinc carbonate sulfate or oxide are all effective as sources of zinc in the treatment or prevention of parakeratosis

Other Minerals

Special situations which bring about increased requirements or dietary imbalances may indicate the need for additional mineral elements Several experiments suggest a need for cobalt by pigs fed an all plant type of basal ration and maintained in dry lot However it is doubtful that cobalt is necessary if the ration is adequate in vitamin B_{12} Evidence is likewise available both for and against the need of certain trace elements especially copper in growing fattening rations in dry lot Sulfur while probably required by swine is provided in ample amounts by the sulfur containing amino acids Inorganic sources of sulfur such as flowers of sulfur are not utilized by pigs

VITAMINS

Vitamins are distributed in small amounts in feeds and are required by swine for health and well being They are unrelated to each other chemically and differ from the structural and energy yielding compounds in the ration in that they are required in small amounts and their role in living processes concerns specific physiological functions Most vitamins exist in more than one form or modification and their distribution in feeds varies Tissue alterations and symptoms due to vitamin deficiencies vary depending on the specific biochemical functions of the vitamin

Dividing the vitamins on the basis of solubility has a usefulness in grouping certain physiological characteristics The fat soluble vitamins are absorbed and partially metabolized with the lipids are stored in relatively large quantities and may be toxic in excessive amounts The water soluble vitamins are absorbed more readily are not stored as well and are seldom toxic even in large amounts

In swine knowledge of the vitamins has come from two main lines of investigation (1) their value in the treatment and prevention of nutritional diseases mainly rickets and pig pellagra (2) the feeding of semi synthetic or purified diets The use of purified diets or rations of known composition initiated during the late 1930 s (Birch et al 1937 Hughes 1938 Wintrobe 1939) has been employed extensively since to demonstrate requirements biochemical changes symptoms and lesions of deficiencies
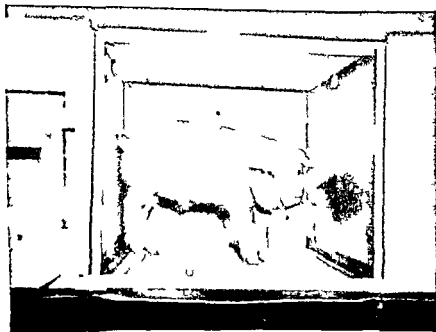
FIG 49 2 — Feeding purified rations to pigs maintained in individual metabolism cages as illustrated has revealed information as to the nutritive requirements of young pigs

Baby pigs from a few days to several weeks of age are used in these studies They are maintained in individual metabolism cages (Fig 49 2) Purified rations make it possible to include, withdraw, or feed at a certain level a specific nutrient with a minimum disturbance to the rest of the ration A wide variety of rations have been successfully employed in these metabolic studies Most of the diets are compounded according to the composition of sow's milk or the requirements of other species, especially the rat Of equal or more importance is maintaining specified environmental temperature and freedom from diseases As a result of these studies a voluminous amount of basic as well as practical information has been made available Swine apparently require some 15 or more vitamins Only those that have been well established and are of practical concern will be considered

Vitamin A

Vitamin A functions mainly in maintaining the structural and functional integrity of epithelial cells, the visual functions of the retina, and growth Epithelial cells with a secretory function are mainly concerned

The ultimate source of all vitamin A is the plant kingdom Cryptoxanthine in yellow corn, carotene in pasture, and good quality roughages are the main precursors or provitamin A's Concentrate feeds, other than corn, are usually devoid of carotene Consequently, swine may encounter vitamin A deficiency when the ration does not include good quality roughage or access to pasture Vitamin A and the carotenoids are highly unsaturated and are easily destroyed by oxidation, which is enhanced by light, heat, rancidity, and oxidants such as the metals Under some conditions yellow corn may lose as much as 50 per cent of its carotene supply in six months It is always important to consider losses in vitamin A or carotene that may result from processing of feeds such as grinding, pelleting, storage, or in curing roughages

The symptoms of vitamin A deficiency

have been reported by numerous workers (Hughes *et al*, 1928, Biester and Murray, 1933 Dunlop 1934 Elder, 1935 Hentges *et al*, 1952a) As might be expected because of the tissues involved, vitamin A deficiency shows a wide variety of symptoms An early symptom in pigs is a tendency to carry the head tilted to one side This is believed to be due to an infection of the inner ear (otitis media) An incoordination — more of a swaying gait — followed by a loss of control and, eventually paralysis of the rear limbs is a common symptom noted by most investigators Pigs also show stiffness in walking lordosis, spasms (Fig 49 3), and extreme restlessness Appetite and rate of gain are apparently not affected Night blindness and minor eye lesions occur late in the deficiency In pregnant gilts resorption of fetuses abortion and birth of dead pigs occur Congenital defects of anophthalmia, cleft palate, and microphthalmia have been noted under experimental conditions (Hale, 1935) and in field cases (Watt and Barlow, 1956)

Vitamin A deficiency in young animals produces a retarded bone growth — even before symptoms are noted This is believed (Wolbach and Bessey, 1942) to produce an overcrowding of the cranial cavity and spinal canal as the central nervous system continues to grow In pigs it causes spasms and paralysis degeneration in portions of the spinal cord, sciatic and femoral nerves (Hughes *et al*, 1928), and increase

FIG 49 3 — Experimentally produced vitamin A-deficient pig showing lordosis and weakness of hind legs. (Courtesy Hentges et al, 1952)

in cerebrospinal pressure (Sorensen *et al*, 1954) In epithelial tissue a deficiency causes atrophy of the epithelial layer, followed by reparative proliferation of the basal cells with growth and differentiation into a stratified keratinizing epithelium

The average level of vitamin A in the blood plasma of normal young pigs 3 weeks to 4 months of age was found (Hentges *et al*, 1952a) to be 23 µg per 100 ml and these values dropped to below 5 µg per 100 ml before visible vitamin A deficiency symptoms were noted

A minimum daily requirement of 25 µg of purified carotene per kilo of body weight has been suggested (Hentges *et al*, 1952b) Swine efficiently convert carotene into vitamin A in the intestinal wall (Swick *et al* 1952) The vitamin A is stored in the liver and Kupffer s cells there is also some storage of vitamin A in the kidneys Apparently large amounts of vitamin A can be stored under some conditions, as it has been reported to take 20 months to deplete the reserves of a sow on a vitamin A deficient diet (Braude *et al*, 1951)

Vitamin D

Vitamin D, also known as the anti rachitic factor, functions in a number of ways in calcium and phosphorus metabolism In addition to having an effect on deposition of calcium and phosphorus in osteoid tissue, it also functions in absorption of calcium from the intestine, main taining specific blood levels of calcium and phosphorus and activating the phosphatase enzyme The two most important forms of vitamin D are vitamins D_2 and D_3 Vitamin D_2 (irradiated ergosterol or calciferol) is produced by exposure of plant sterols to ultraviolet light and is present in sun cured roughages and irradiated yeast. Vitamin D_3 (irradiated 7-dehydrocholesterol) is produced in the body on irradiation It is also present in fish oils and in animal products Swine use both types of vitamin D with equal efficiency

In swine, vitamin D deficiency occurs in the northern climates where there is a minimum exposure to ultraviolet rays

The symptoms of deficiency (Johnson and Palmer, 1939, 1941) are loss of appetite, unthrifty appearance, rough haircoat and unusual lameness Blood plasma calcium values decrease from normal values of 10 to 12 mg per 100 ml to 6 mg or below At these levels tetany is observed Pigs with rickets can be cured on exposure to the sun for 45 minutes a day for two weeks Colored breeds of pigs are more susceptible to a deficiency than the white breeds

Excessive amounts of vitamin D may produce a toxicity which is characterized by a hypercalcemia with calcium deposits in the large blood vessels, especially the aorta and heart, and in the kidneys

Vitamin E

It is becoming increasingly apparent that the physiological effects of vitamin E depend on its antioxidant properties There are several biologically active vitamin E or tocopherol compounds Of these, alpha tocopherol is the most active Vitamin E deficient rations cause disturbances in reproduction in laboratory animals, and various forms of muscular dystrophy, because it is required in the metabolism of skeletal and cardiac musculature

It has generally been assumed that a vitamin E deficiency will not occur in swine However, investigations indicate that swine may be susceptible to a vitamin E deficiency under certain conditions Sows experimentally fed rations low in vitamin E reproduced poorly, apparently as a result of death of embryos rather than of interference with ovulation and implantation (Adamstone *et al*, 1949) Pigs from the deficient sows were continued on vitamin E-deficient diets and developed muscular incoordination due to degeneration and necrosis of the muscle fibers In pigs, disturbances were produced when experimental rations containing various amounts of unsaturated fats were fed The pigs developed a yellow to yellowish brown pigmentation of the adipose tissue, died suddenly, and showed lesions of acute hemorrhagic liver necrosis (Gorham *et al*, 1951, Davis and Gorham, 1951, Hove and Sei

bold, 1955) The symptoms were reduced or prevented with vitamin E

A naturally occurring problem in pigs fed rations rich in cod liver oil was studied in Scotland (Garton and Naftalin, 1953) This condition was characterized by edema of the neck and abdomen and resembled the exudative condition produced in chickens fed high fat diets deficient in vitamin E Likewise, in Sweden and North European countries a frequently observed problem occurs in pigs 3 to 6 weeks old that is believed due to a deficiency of vitamin E and the sulfur containing amino acids (Obel, 1953) The characteristic symptoms are muscular weakness anemia and sudden death The outstanding pathology is an extensive necrosis of the liver Also, noted less frequently is ulceration of the stomach and colon, waxy degeneration of the skeletal muscles, hemorrhagic lymph nodes, and pulmonary edema

Vitamin E is widely distributed in feeds Whole cereals, especially the germ part, and green forages are good sources The wide variety of feeds and feeding methods employed in swine operations would seem to indicate that a vitamin E deficiency may occur when large amounts of unsaturated fats are included in rations and thus increase vitamin E requirements

Vitamin K

There are several quinone compounds that have vitamin K activity The most active is 2 methyl 3 phytyl 1, 4 naphthoquinone In vitamin K deficiency the prothrombin level of the blood is decreased

In swine there is no evidence that a vitamin K deficiency is likely to be encountered under practical feeding and management operations Accidental poisoning of pigs with Warfarin, a derivative of dicoumarin and an antagonist to vitamin K, has been observed to produce extensive subcutaneous hemorrhages on bruising (Clark, 1951)

Thiamine (Vitamin B₁)

Thiamine functions in carbohydrate metabolism In a deficiency there is in complete carbohydrate metabolism, and pyruvic acid accumulates in the tissues The basis for most of the symptoms manifested are believed due to this disturbance High fat diets decrease the requirements for thiamine

Using experimental type diets the need for thiamine by young pigs was first reported by Hughes (1940b) and Van Etten et al (1940) The symptoms of a deficiency are marked inappetence, poor growth vomition diarrhea cyanosis of the skin and mucous membranes, and sudden death In more detailed studies Wintrobe et al (1942a 1943a), Follis et al (1913), and Miller et al (1955) found cardiac dilation, slowing of the heart necrosis of the cardiac muscle fibers and pronounced electrocardiographic changes Nerve lesions described for this deficiency in the early work have not been confirmed

Thiamine is not stable to heat, especially in the presence of alkali It is widely distributed in feeds such as the cereal grains, animal by products and brewers yeast It is unlikely that an uncomplicated thiamine deficiency occurs in swine except under experimental conditions

Riboflavin (Vitamin B₂)

Riboflavin is required by all living cells It is a constituent of several enzymes and functions in oxidative processes whereby food energy is made available to the cell A deficiency affects especially the tissues of ectodermal origin A wide variety of deficiency symptoms have been reported in various species, and this would not be unexpected in view of the basic function of riboflavin in cellular metabolism Cataracts of the eyes and dermatitis of the skin have been noted in most species

In swine, the symptoms and lesions have been reported by Hughes (1910a), Wintrobe et al (1911), Lehrer and Wiese (1952), and Miller et al (1951) They are slow growth, vomition, cataracts abnormal stiffness and gait, eruption, scaling and ulceration of the skin, and alopecia A normocytic anemia and myelinic degen-

eration of nerve tissue have been reported by several workers In sows, a deficiency causes a poor reproduction and lactation performance (Miller et al, 1953) In pigs, the requirements are increased in a cold environment (Mitchell et al, 1950)

Riboflavin is a water soluble yellowish pigment It is stable to heat but is destroyed readily by visible or ultraviolet light The green leafy forages are good sources of riboflavin In contrast to thiamine the cereals are rather low in this vitamin

Pantothenic Acid

Pantothenic acid functions as a coenzyme in many biochemical reactions involving acetic acid and closely related two carbon fragments Thus, it is involved in the metabolism and synthesis of fats, carbohydrates, and many other compounds

Hughes and Ittner (1912), Wintrobe and associates (1912b, 1943b), and Wiese et al (1951) studied the symptoms and tissue changes of a pantothenic acid deficiency in young pigs The symptoms observed are inappetence, poor growth, diarrhea, coughing, loss of hair, and locomotor incoordination or 'goose stepping' (Fig 49 4) The characteristic necropsy lesion is an involvement of the intestine, especially edema congestion and inflammation of the colon Histological studies of the colon

FIG 49 4—Symptoms of "goose stepping' and locomotor incoordination experimentally produced by feeding a pantothenic acid low ration (Courtesy Michigan Agricultural Experiment Station)

show degenerative changes, lymphocytic infiltration, and hyperemia of the lamina propria In nerve tissue, degeneration of the peripheral nerves, posterior root ganglia, posterior roots, and the funiculi of the spinal cord are evident Detailed studies by Sharma et al (1952) confirm the earlier findings, especially the histopathological changes in the gastrointestinal tract Reproduction lactation performance is upset when sows are fed pantothenic acid deficient rations (Ullrey et al, 1955)

Pantothenic acid is stable to heat and light and is available as the calcium salt, calcium pantothenate While it is widely distributed in swine feeds, concentrated natural sources are more limited than some of the other B complex vitamins Also symptoms of "goose stepping," which is a characteristic symptom of pantothenic acid deficiency, have been observed under field conditions (Elder, 1935, Doyle, 1937) Thus a deficiency of pantothenic acid in pigs fed natural feedstuffs might be encountered in the field (Luecke et al, 1950b)

Nicotinic Acid (Niacin)

This was the first B complex vitamin demonstrated to be indispensable for swine Chick et al (1938) in England discovered that a 'pellagra producing' ration, of which corn was the chief ingredient, produced a severe diarrhea and dermatitis in pigs Nicotinic acid brought about a rapid and dramatic cure The disease in pigs has been referred to as 'pig pellagra,' after its counterpart of the disease in the human

Nicotinic acid is a component of several enzyme systems in oxidation reduction reactions The amide of nicotinic acid is the physiological active compound Thus, a more proper name for the vitamin is nicotinamide It is quite resistant to heat and therefore stable in feeds

Reports from other laboratories soon followed the British report confirming the initial observations and extending additional information on the importance of niacin in swine nutrition (Madison et al, 1939, Davis et al, 1910, Hughes, 1913).

A deficiency of nicotinic acid in pigs is characterized by loss of appetite emaciation severe diarrhea dermatitis nervous disorders and anemia At necropsy marked pathological changes are noted in the intestinal tract (Dunne *et al*, 1949) The intestinal wall, especially the colon and cecum is thickened friable and may feel corky or pulpy The mucosa lining may be discolored and the colon contents so firmly adhered that they are difficult to wash off with water (Fig 49 5) On microscopic examination the mucosae of the colon and cecum show severe mucinous degeneration The goblet cells are distended with secretory fluid and necrosis is evident in most areas of mucinous degeneration The entire colon may exhibit a chronic inflammatory process with macrophagic lymphocytic and neutrophilic infiltration Hemorrhagic lesions and congestion may also be present in the mucous lining of the stomach and small intestine The mesenteric lymph nodes are usually enlarged and edematous

NIACIN AND TRYPTOPHAN

In 1945 Wintrobe and co workers (1945) reported that a nicotinic acid deficiency could not be produced in young pigs fed a ration containing 26 per cent casein In other species it was noted that tryptophan which is low in corn and in pellagra producing diets would overcome a nicotinic acid deficiency Thus it was soon established (Luecke *et al*, 1948 Powick *et al* 1948) that a similar relationship exists in the pig since tryptophan an amino acid served as a precursor for niacin in the body Niacin is not converted to tryptophan In formulating swine rations assurance should be made that niacin supplies are adequate so that the more expensive and often limiting supply of tryptophan is used for protein synthesis and not for synthesis of niacin

NIACIN AND NECROTIC ENTERITIS

In 1940 Davis and co-workers (Davis and Freeman 1940 Davis *et al* 1940) concluded that nicotinic acid was of considerable value in the prevention and cure of necrotic enteritis an ill-defined disease of pigs believed to be of infectious origin They believed the disease to be secondary to a deficiency of nicotinic acid This disease is very prevalent in most swine raising areas and there was considerable support from practicing veterinarians that nicotinic acid was valuable in treating disturbances in pigs characterized by unthriftiness poor growth and digestive disorders

Workers from Wisconsin (Fargo *et al.*, 1941) and Ohio (Edgington *et al.*, 1942) could not confirm the observation that nicotinic acid was of value in preventing necrotic enteritis in pigs. The Ohio workers exposed pigs to *Salmonella choleraesuis*, the organism commonly associated as a primary pathogen in necrotic enteritis, and concluded that the protective value of nicotinic acid against *S. choleraesuis* infection was not sufficient to encourage its use as a specific preventive or curative measure. In further work by Davis *et al.* (1943), they noted that nicotinic acid did not prevent the pigs from reacting to *S. choleraesuis* infection, but following initial infection it was effective in promoting rapid recovery.

It would seem that in pigs there are two separate conditions that appear similar in clinical and pathological manifestations. One is a nutritional deficiency, pig pellagra produced by rations deficient in niacin and low in protein. The other is a specific infection involving the digestive tract. Niacin is not of value in *preventing* the infection, however, due to the excessive losses of nutrients, especially protein and vitamins due to diarrhea, it may be of value in restoring the pigs to recovery. It would seem that niacin would have a dual role in digestive disturbances (1) that of the specific functions of nicotinamide (2) in sparing tryptophan for synthesis of tissue protein and not conversion to niacin

AVAILABILITY OF NIACIN IN CORN

Evidence has persisted that corn has some positive or pellagragenic effect in the production of pellagra. High-corn low protein rations were used to produce niacin deficiency in pigs. A wide variation in the severity of symptoms has been noted in pigs fed the same niacin-deficient ration (Burroughs *et al.*, 1950). In addition to the tryptophan deficiency in corn and the tryptophan niacin interrelationship another relationship noted by Kodicek *et al.* (1956) is that niacin in corn and perhaps other cereals is in an alkali labile bound form unavailable to the pig. These

workers produced a niacin deficiency in pigs on a high corn ration and then cured it by either supplementing niacin or subjecting the ration to weak alkaline hydrolysis.

This latter observation supports recommendations (Hofferd, 1936; Wilson, 1940; Graham *et al.*, 1945) as to the value of feeding rations composed of cereals (other than corn), especially oats, that have been soaked in an alkaline medium as supportive treatment for enteric infections in pigs. Besides supplying additional niacin, such a ration would also supply additional amounts of some of the limiting amino acids (tryptophan and lysine), minerals, and vitamins (Whitehair *et al.*, 1948b)

Pyridoxine (Vitamin B_6)

Pyridoxine functions as an enzyme in protein metabolism, especially in protein synthesis.

A deficiency produced experimentally in swine has been described by Hughes and Squibb (1942), Wintrobe *et al.* (1943c), and Lehrer *et al.* (1951). It is characterized in pigs by poor growth, diarrhea, a severe microcytic hypochromic anemia, convulsions, ataxia, and fatty infiltration of the liver. Preceding the epileptiform convulsions the pigs are usually excited and nervous. On histological examination there is evidence of demyelination of the brachial, sciatic and peripheral nerves. The anemia is believed to be due to a disturbance in the utilization of iron.

A deficiency of pyridoxine in pigs is somewhat difficult to produce. It is widely distributed in substantial amounts in feeds and a deficiency under usual swine raising operations would not be expected

Cyanocobalamin (Vitamin B_{12})

It was established in the early 1940's that when swine were maintained under dry lot conditions and fed an all plant type of ration, a factor found in fish meal, liver, meat scraps, whey, milk, and other animal products was required for optimum growth, reproduction, and lactation. This

factor became known as the animal protein factor (APF)

In 1947 the factor in liver which was effective in treating pernicious anemia in the human was isolated and named vitamin B_{12} Shortly thereafter it was established that this was the same as the long sought after animal protein factor In 1955 the chemical structure was determined and the chemical name, cyanocobalamin applied Several closely related compounds have vitamin B_{12} activity

The specific function of vitamin B_{12} has not been determined In the human it is required in erythropoiesis to prevent megaloblastic anemia and nervous disturbances associated with pernicious anemia It functions, probably as an enzyme, in the synthesis of nucleic acids and methyl groups Evidence that it functions in transmethylation and in protein utilization has largely been disproved (Henry and Kon, 1956) Vitamin B_{12} is synthesized in the normal intestinal tract — presumably in the colon Apparently there is a wide range of variation in the intestinal absorption (Jerzy Glass *et al*, 1956) Under experimental conditions a vitamin B_{12} deficiency has been produced in the pig (Neumann *et al*, 1950, Baumedel *et al*, 1951) However, the importance of vitamin B_{12} in practical swine nutrition remains uncertain Numerous workers (Nesheim *et al*, 1950 Anderson and Hogan, 1950, Vohs *et al*, 1951, Catron *et al*, 1952, Luecke *et al*, 1950a, Robison, 1953) have noted a response in pigs maintained in dry lot and fed basal rations composed mainly of corn and soybean meal Other workers (Colby and Ensminger, 1950, Blight *et al*, 1952 Burnside *et al*, 1951, Hendebrecht *et al*, 1949, Meade, 1956b) have not observed any response to vitamin B_{12} supplementation Burnside and associates maintained pigs in dry lot and fed a basal ration of corn, soybean meal, and alfalfa meal They found vitamin B_{12} of little or no help in increasing the rate of gain, feed efficiency, hemoglobin, plasma protein, or digestibility. The discrepancies between various experiments probably depend on the type of ration fed and the previous storage of vitamin B_{12}, the health status of the digestive tract intestinal synthesis and whether or not the pigs have access to their feces

In the pig the requirement has been given as less than 5 μg per lb of ration In the human the requirement for maintenance and good health is 1 μg or less a day and a deficiency is nearly always secondary to disease of the alimentary tract" (Witts, 1956) For sows during reproduction lactation adding a small amount of animal protein would be cheap insurance against a vitamin B_{12} deficiency

Fermentation residues are widely used as a practical source of vitamin B_{12} It was observed in 1949 that the response in pigs to certain residues was greater than could be explained on the basis of vitamin B_{12} alone The added response was found to be due to residues of antibiotics in the fermentation products

Pteroylglutamic Acid (Folic acid)

Folic acid is required in erythropoiesis The physiologically active forms of the vitamin are referred to as folinic acid and citrovorum factor Using synthetic type rations and a folic acid antagonist, a deficiency has been produced in the pig (Johnson *et al*, 1948). It is characterized by poor growth, weakness, diarrhea, and a normocytic anemia Supplementing even simple rations with folic acid has not improved growth, reproduction, or lactation and a deficiency under practical feeding operations would not be expected Folic acid is widely distributed in animal and plant products, especially green leafy feeds

Biotin

A biotin deficiency has been produced in pigs by feeding a ration composed of 54 per cent dried egg white to tie up the biotin in an insoluble complex (Cunha *et al*, 1946) It has also been produced more recently, using a synthetic type of ration (Lehrer *et al*, 1952) Deficiency symptoms include alopecia dermatosis, and ulceration of the skin, spasticity of the hind legs, transverse cracking and bleed

ing of the feet, and inflammation of the mucous membrane of the mouth Biotin is synthesized in the intestinal tract. It is widely distributed in feeds, and there is no evidence that a deficiency may occur under farm conditions

Choline

Choline is a component of lecithin, a phospholipid, and is concerned in fat assimilation and transport — a deficiency produces a fatty liver (Johnson and James, 1918) Choline is interrelated with methionine, cystine, and betaine, and these compounds can to a certain extent replace each other in rations It is unlikely that a deficiency would occur using practical swine rations

Additional Growth Factors

Additional growth factors, such as inositol and para aminobenzoic acid, have

been identified, but no need by swine has been established Vitamin C is not required by swine (Hughes *et al*, 1928, Grummer *et al*, 1948) From time to time additional factors from crude sources such as "whey factor," "grass juice factor," 'fish solubles factor,' 'alfalfa factor,' etc., are proposed Additional research is required to establish the importance of both known and unidentified nutrients for swine, during reproduction and lactation Numerous investigators have demonstrated nutritional inadequacies in rations fed to sows during reproduction and lactation (Hogan and McRoberts, 1940, Ross *et al*, 1941, Gwatkin and Plummer, 1918, Furbanks *et al*, 1915, McElroy and Draper, 1950) Additional information is likewise needed as to the interrelationships not only between various nutrients but also between stress factors, diseases, and environment

REFERENCES

Armstone, F B Krider J L And James M F 1949 Response of swine to vitamin A deficient rations Ann New York Acad Sci 52 260

Anderson G C and Hogan, A G 1950 Requirement of the pig for vitamin B_{12} Jour Nutrit 40 13

Andrews F N Shrimsbury, C L Harler, C Vestal C M and Doyle, L P 1948 Iodine deficiency in newborn sheep and swine Jour Anim Sci 7 298

Balbert W R Hoertens A B Tickes J C Jr. and Underkofler L A 1951 Pig nutrition selection of diet for studies of vitamin B_{12} depletion using unsuckled baby pigs Jour Agr and Food Chem 2 163

Becker D E, Lasster J W., Terrill S W and Norton H W 1951a Levels of protein in practical rations for the pig Jour Anim Sci 13 611

——— Ullrey D L Terrill, S W and Notzold, R A 1951b Failure of the newborn pig to utilize dietary sucrose Science 1 0 345

Biester H E, and Murray C 1933 Posterior paralysis in young pigs Iowa Vet 1 5

Birch J W Chick H and Martin C J 1937 Experiments with pigs on a pellagra producing diet Biochem Jour 31 2065

Birch J G, King J A and Ellis N R 1952 Effect of vitamin B_{12} and aureomycin concentrates on the growing rate of unthrifty weanling pigs Jour Anim Sci 11 92

Bohstedt G 1910 Mineral and vitamin requirements of pigs. Ohio Agr Exp Sta Bull 395

——— 1953 The mineral requirements of pigs. Proc Amer Soc Anim Prod 137

Bradley R Ross S K Mitchell K G and Thomason S Y 1951 Some observations on vitamin A requirements of pigs. Vet Rec. 63 671

Bryant J B 1954 Some methods employed in my swine practice Cornell Vet 45 61

Burnside J F Cremmer R H Lutitius P H and Bohstedt C 1951 The influence of crystalline aureomycin and Vitamin B_{12} on the protein utilization of growing fattening swine Jour Anim Sci 13 161

Burroughs W., Lewington R H Robison W., and Bethke R M 1950 Niacin deficiency and its effect in growing pigs Jour Nutrit 41 51

Cannon P R 1948 Some particular consequences of Protein and Amino Acid Deficiencies Charles C Thomas Springfield Ill

——— 1949 Recent Advances in Nutrition With Particular Reference to Protein Metabolism Univ Kans Press Lawrence Kans

Catron D V, Richardson, D, Underkofler, L A, Maddock, H M and Friedland, W C 1952 Vitamin B_{12} requirement of weanling pigs II Jour Nutrit 47 161

Chick, H Macray T E, Martin A J P, and Martin C J. 1938 Curative action of nicotinic acid on pigs suffering from the effects of a diet consisting largely of maize Biochem Jour 32 10

CLARK, S T 1951 A case of Warfarin poisoning in young pigs Vet Rec 66 78

COLBY, R W, AND ENSMINGER, M E 1950 Effect of vitamin B_{12} on the growing pig Jour Anim Sci 9 90

CUNHA, T J, LINDLEY, D C, AND ENSMINGER M E 1946 Biotin deficiency syndrome in pigs fed desiccated egg white Jour Anim Sci 5 219

DAVIS, C. L. AND GORHAM, J R 1951 The pathology of experimental and natural cases of 'yellow fat' disease in swine Amer Jour Vet Res 15 55

DAVIS, G K. AND FREEMAN, V A 1940 Studies upon the relation of nutrition to the development of necrotic enteritis in young pigs fed massive doses of S choleraesus Proc Amer Soc. Anim Prod 316

———, AND MADSEN, L L 1940 The relation of nutrition to the development of necrotic enteritis in swine Mich Agr Exp Sta Tech Bull 170

———, HALE, E B AND FREEMAN V A 1943 Response of pigs given large doses of Salmonella choleraesus to sulfaguanidine, nicotinic acid thiamine and pyridoxine Jour Anim Sci 2 138

DOYLE, L P 1937 Posterior paralysis in swine Jour Amer Vet Med Assn 90 606

———, MATHEWS, F P, AND WHITING R A 1927 Anemia in young pigs Ind Agr Exp Sta Bull 313

DUNLOP, G 1931 Paralysis and avitaminosis A in swine Jour Agr Sci 21 435

DUNNE, H W, LUECKE, R W MCMILLEN, W N GRAY, M L AND THORP, F Jr. 1949 The pathology of niacin deficiency in swine Amer Jour Vet Res 10 351

EDGINGTON, B H ROBISON, W L BURROUGHS W, AND BETHKE R M 1942 Tests with nicotinic acid for the prevention of infectious swine enteritis Jour Amer Vet Med Assn 101 103

EGGERT, R G, BRINEGAR, M J AND ANDERSON, C R 1953 Delayed protein supplementation of corn diets for growing swine Jour Nutrit 50 469

ELDER, C 1935 Avitaminosis A in swine Jour Amer Vet Med Assn 87 22

EVVARD, J M CULBERTSON, C C HAMMOND, W E AND WALLACE, Q W 1925 White gold in the swine kingdom Iowa Agr Exp Sta Leaflet 7

FAIRBANKS, B W, KRIDER, J L, AND CARROLL, W E 1945 Effect of diet on gestation lactation performance of sows Jour Anim Sci 4 410

FARGO, J M, WHITEHAIR C K, AND BOHSTEDT G 1941 What can be done about necro? Wis Agr Exp Sta Bull 451

FOLLIS, R H Jr., DAY, H G AND MCCOLLUM E B 1941 Histological studies of the tissues of rats fed a diet extremely low in zinc Jour Nutrit 22 223

———, MILLER, M H WINTROBE, M M AND STEIN, H J 1943 Development of myocardial necrosis and absence of nerve degeneration in thiamine deficiency in pigs Amer Jour Path 19 341

FRADKIN, W Z 1953 The dietary treatment of diarrheal diseases Amer Jour Digest Dis 20 208

GARTON, G A, AND NAFTALIN, J M 1953 Massive oedema (exudative diathesis) in pigs fed on a diet rich in cod liver oil Vet Rec 65 262

GORHAM, J R., BOE, N AND BAKER G A 1951 Experimental Yellow Fat disease in pigs Cornell Vet 41 332

GRAHAM, R. PETERSON E H MORRILL, C C HARDENBROOK H J WHITMORE G E AND BEAMER, P D 1945 Studies on porcine enteritis I Sulfathalidine therapy in treatment of natural outbreaks Jour Amer Vet Med Assn 106 7

GRUMMER, R. H, BENTLEY, O G, PHILLIPS, P H AND BOHSTEDT, G 1950 The role of manganese in growth reproduction and lactation of swine Jour Anim Sci 9 170

———, WHITEHAIR C K, BOHSTEDT, G, AND PHILLIPS P H 1948 Vitamin A vitamin C and niacin levels in the blood of swine Jour Anim Sci 7 222

GWATKIN, R AND PLUMMER, P J G 1948 Mortality in young pigs Canad Jour Comp Med 12 116

HALE, F 1935 The relation of vitamin A to anophthalmus in pigs Amer Jour Ophthal 18 1087

HAMILTON, T S, HUNT, G E., MITCHELL H H, AND CARROLL, W E 1930 The production and cure of nutritional anemia in suckling pigs Jour Agr Res 40 927

HANSON, L E AND FERRIN, E T 1955 The value of urea in a low protein ration for weanling pigs Jour Anim Sci 14 43

——— PRITCHARD W R REIFELD C E, PERMAN V SAUTTER J H AND SCHULTZE M O 1956 Studies on trichloroethylene extracted feeds. IX Experiments with swine fed trichloro ethylene extracted soybean oil meal Jour Anim Sci 15 368

HART, E B ELVEHJEM C A STEENBOCK, H BOHSTEDT, G AND FARGO J M 1929 Anemia in suckling pigs Wis Agr Exp Sta Bull 409

HASTINGS, C C 1955 Milk fever of sows No Amer Vet 36 102

HEIDENREICH, C A, ROSS O B., MACVICAR, R W., AND WHITEHAIR, C K 1919 The effect of iodine fish soulubles and vitamins A and B_{12} supplementation on the reproduction and lactation performance of sows fed plant rations Jour Anim Sci 8 621

HENRY, K M, AND KON, S K 1956 Vitamin B_{12} and protein metabolism Brit Jour Nutr 10 39

Hentges J F Jr Grummer R H Phillips P H and Bohstedt G 1952b The minimum requirement of young pigs for a purified source of carotene Jour Anim Sci 11 266

—— —— and Sorenson D K 1952a Experimental avitaminosis A in young pigs Jour Amer Vet Med Assn 120 213

Hillier J C and Whitehair C K 1952 The effects of adding soybean meal B vitamins and alfalfa meal to a corn mineral and antibiotic diet for young pigs Okla Agr Exp Sta Misc Publ MP 27 38

Hofferd R M 1936 Swine dysentery in Iowa from a field standpoint Jour Amer Vet Med Assn 88 299

Hogan A G and McRoberts V F 1940 Vitamin deficiencies in a ration for brood sows Proc Amer Soc Anim Prod 139

Hove E L and Seibold H R 1955 Liver necrosis and altered fat composition in vitamin E deficient swine Jour Nutrit 56 173

Hughes E H 1938 The vitamin B complex as related to growth and metabolism in the pig Hilgardia 11 595

—— 1940a The minimum requirement of riboflavin for the growing pig Jour Nutrit 20 233

—— 1940b The minimum requirement of thiamin for the growing pig Jour Nutrit 20 239

—— 1942 The potassium requirement of growing pigs Jour Agr Res 64 189

—— 1943 The minimum requirement of nicotinic acid for the growing pig Jour Anim Sci 2 23

—— and Ettner N R 1942 The minimum requirement of pantothenic acid for the growing pig Jour Anim Sci 1 116

—— and Squibb R L 1942 Vitamin B₆ in swine nutrition Jour Anim Sci 1 320

Hughes J S Aubel C E and Lienhardt H F 1928 The importance of vitamin A and vitamin C in the ration of swine Kans Agr Exp Sta Tech Bull 23

Jerzy Glass G B Goldbloom A A Boyd L J Laughton R Rosen S and Rich M 1956 Intestinal absorption and hepatic uptake of radioactive vitamin B₁₂ in various age groups and the effect of intrinsic factor preparations Amer Jour Clin Nutr 4 194

Johnson B C and James M F 1948 Choline deficiency in the baby pig Jour Nutrit 36 339

—— and Krider J L 1948 Raising newborn pigs to weaning age on a synthetic diet with attempt to produce a pteroylglutamic acid deficiency Jour Anim Sci 7 486

Johnson D W and Palmer L S 1939 Individual and breed variations in pigs on rations devoid of vitamin D Jour Agr Res 58 929

—— and —— 1941 Meeting the vitamin D requirements of pigs with alfalfa hay and winter sunshine Jour Agr Res 63 639

Johnson S R 1944 Studies with swine on low manganese rations of natural foodstuffs Jour Anim Sci 3 136

Jones J H Foster C Henke W and Alexander D 1946 Dietary deficiencies and poliomyelitis Effects of low protein and of low tryptophan diets on the response of mice to the Lansing strain of poliomyelitis virus Arch Biochem 11 481

Keith T B Miller R C Thorp W T S and McCarty M A 1942 Nutritional deficiencies of a concentrate mixture composed of corn tankage soybean oil meal and alfalfa meal for growing pigs Jour Anim Sci 1 120

Kernkamp H C H 1945 A study of a disease of the bones and joints of swine Minn Agr Exp Sta Tech Bull 31

—— 1944 Diseases of swine due to nutritive deficiencies Jour Amer Vet Med Assn 99 373

—— 1945 Gastroenteric disease in swine Jour Amer Vet Med Assn 106 1

Kodicek E Braude A Kon S K and Mitchell K G 1956 The effect of alkaline hydrolysis of maize on the availability of its nicotinic acid to the pig Brit Jour Nutr 10 51

Lehrer W J Jr and Wiese A C 1952 Riboflavin deficiency in baby pigs Jour Anim Sci 11 215

—— and Moore L R 1952 Biotin deficiency in suckling pigs Jour Nutrit 17 203

—— and Ensminger M E 1951 Pyridoxine deficiency in baby pigs Jour Anim Sci 10 65

Leitch I and Thomson J S 1944 The water economy of farm animals Nutr Abst and Revs 14 197

Lewis P K Jr Hoekstra W G, Grummer R H and Phillips P H 1956 The effect of certain nutritional factors including calcium phosphorus and zinc on parakeratosis in swine Jour Anim Sci 15 741

Loeffel W J Thalman R R, Olson F C and Olson F A 1951 Studies of rickets in swine Nebr Agr Exp Sta Res Bull 58

Luecke R W, Hoeffer J A, Brammell, W S, and Thorp F Jr 1956 Mineral interrelationships of swine Jour Anim Sci 15 347

—— McMillen W N and Thorp F Jr 1950a The effect of vitamin B₁₂ animal protein factor and streptomycin on the growth of young pigs Arch Biochem 26 326

—— —— and —— 1950b Further studies of pantothenic acid deficiency in weanling pigs Jour Anim Sci 9 78

————, ————, ————, AND TULL C 1948 Further studies on the relationship of nicotinic acid, tryptophane and protein in the nutrition of the pig Jour Nutrit 36 417

————, THORP, F JR, McMILLEN W N DUNNE H W AND STAFSETH H J 1919 A study of vitamin B deficiencies in pigs raised on farms Mich Agr Exp Sta Tech Bull 211

McELROY, L W., AND DRAPER, H H 1950 Effect of inadequate brood sow rations on the pre natal and postnatal development of the progeny Sci Agr 30 172

McGOWAN, J P., AND CRICHTON, A 1923 On the effect of deficiency of iron in the diets of pigs Biochem 17 204

MADISON, L C MILLER, R C, AND KEITH T B 1939 Nicotinic acid in swine nutrition Science 89 490

MARRIOTT, H L 1950 Water and Salt Depletion A Monograph in Amer Lec in Phys Charles C Thomas, Springfield

MAYNARD, L A., AND LOOSLI, J K 1956 Animal Nutrition 4th ed McGraw Hill Book Co Inc, New York

MEADE, R J 1956a The influence of tryptophan methionine and lysine supplementation of a corn soybean oil meal diet on nitrogen balance of growing swine Jour Anim Sci 15 288

———— 1956b The influence of protein content of the diet and of chlortetracycline and/or vitamin B₁₂ supplementation upon performance of growing fattening pigs Jour Anim Sci 15 297

MILLER, C O, ELLIS, N R, STEVENSON J W AND DAVEY R 1953 The riboflavin requirements of swine for reproduction Jour Nutrit 51 163

MILLER, E R JOHNSTON, R L HOEFER J A AND LUECKE, R W 1951 The riboflavin require ment of the baby pig Jour Nutrit 52 405

———— SCHMIDT, D A HOEFER, J A AND LUECKE R W 1955 The thiamine requirement of the baby pig Jour Nutrit 56 423

MILLER, R C., KEITH, T B., McCARTY, M A AND THORP W T S 1940 Manganese as a possible factor influencing the occurrence of lameness in pigs Proc Soc Exper Biol and Med 45 59

MINER, R W 1955 Nutrition in infections Ann New York Acad Sci 63 145

MITCHELL, H H 1929 Mineral deficiencies in swine rations Jour Amer Vet Med Assn 74 651

———— JOHNSON, B C HAMILTON, T S AND HAINES, W T 1950 The riboflavin requirement of the growing pig at two environmental temperatures Jour Nutrit 41 317

MORRISON, F B 1956 Feeds and Feeding 22 ed Morrison Publishing Co Ithaca N Y

NATIONAL RESEARCH COUNCIL 1953 Nutrient Requirements for Swine Washington D C

NEHER, G M., DOYLE, L P., THRASHER, D M AND PLUMLEE, M P 1956 Radiographic and his topathological findings in the bones of swine deficient in manganese Amer Jour Vet Res 17 121

NESHEIM, R O KRIDER, J L AND JOHNSON, B C 1950 The quantitative crystalline vitamin B₁ requirements of the baby pig Arch Biochem 27 240

NEUMANN, A L., JOHNSON, B C AND THIERSCH J B 1950 Crystalline vitamin B₁₂ in the nu trition of the baby pig Jour Nutrit 40 403

OBEL, A L 1953 Studies on the morphology and etiology of so called toxic liver dystrophy (Hepatosis diaetetica) in swine Acta Path in Microbiol Scand Suppl 94 1

PLUMLEE, M P., THRASHER, D M., BEESON, W M AND ANDREWS, F N 1956 The effects of a manganese deficiency upon the growth development and reproduction in swine Jour Anim Sci 15 353

POWICK, W C., ELLIS, N R., AND DALE, C N 1918 Relationship of tryptophan to nicotinic acid in the feeding of growing pigs Jour Anim Sci 7 228

REBER, E F., AND WHITEHAIR, C K 1955 The effect of transmissible gastroenteritis on the me tabolism of baby pigs Amer Jour Vet Res 16 116

ROBISON, W L 1953 Vitamin B₁₂ supplements for growing and fattening pigs Ohio Agr Exp Sta Res Bull 729

ROSS, O B PHILLIPS, P H, BOHSTEDT, G, AND CUNHA, T J 1944 Congenital malformations syndactylism talipes and paralysis agitans of nutritional origin in swine Jour Anim Sci 3 406

SHARMA, G L., JOHNSTON, R L., LUECKE R W., HOEFER, J A, GRAY, M L., AND THORP, F JR 1952 A study of the pathology of the intestine and other organs of weanling pigs when fed a ration of natural feedstuffs low in pantothenic acid Amer Jour Vet Res 13 294

SLATTER E E 1955 Mild iodine deficiency and losses of newborn pigs Jour Amer Vet Med Assn 127 149

SORENSEN, D K., KOWALCZYK, T AND HENTGES, J F., JR 1954 Cerebrospinal fluid pressure of nor mal and vitamin A deficient swine as determined by a lumbar puncture method Amer Jour Vet Res 15 258

SOSKIN S AND LEVINE, R 1952 Carbohydrate Metabolism Univ Chicago Press Chicago

STEENBOCK, T L 1942 Problems in swine practice Proc U S Livestock San Assn 37

STEVENSON, J W, AND EARLE, I P. 1956 Studies on parakeratosis in swine Jour Anim Sci 15 1036

Swick, R W, Grummer, R H, and Baumann, C A 1952 The effect of thyroid on carotenoid metabolism in swine Jour Anim Sci 11 273

Theiler A, Dutoit, P J, and Malan, A I 1937 Calcium and phosphorus in the nutrition of the growing pig Onderstepoort Jour Vet Sci and Anim Ind 9 127

Truax E R 1911 Swine practice Jour Amer Vet Med Assn 98 206

Ullrey D E, Becker, D E, Terrill, S W, and Notzold, R A 1955 Dietary levels of pantothenic acid and reproductive performance of female swine Jour. Nutrit 57 401

Van Etten, C H Ellis, N R, and Madsen, L L 1940 Studies on thiamine requirements of young swine Jour. Nutrit 20 607

Voris, R L., Maddock, H M, Catron, D V, and Culbertson, C C 1951 Vitamin B₁₂, APF concentrate and dried whey for growing fattening pigs Jour Anim Sci 10 42

Watt J A and Barlow, R M 1956 Microphthalmia in piglets with avitaminois A as the probable cause Vet Rec 68 780

Welch H 1928 Goiter in farm animals Mont Agr Exp Sta Bull 214

Whitehair, C K 1951 The relation of nutrition to digestive disturbances in swine Proc Amer Vet Med Assn, Milwaukee Wis

——— 1953 Unpublished data Animal Husbandry Dept Oklahoma A & M, Stillwater, Okla

———, Grummer, R H, Phillips, P H, Boisteot, G, and McNutt, S H 1948a Gastro enteritis in pigs Cornell Vet 38 23

———, Slitzer, R. R., Boisteot, G, and Phillips, P H 1948b Tryptophan in digestive disturbances in swine Jour Amer Vet Med Assn 113 475

——— and MacVicar R W 1952 The value of amino acids supplemented to a low protein, all plant ration for swine Okla Agr Exp Sta Misc Pub MP 27 1952

Wiese, A. C, Lehrer, W P, Moore, P R, Pahnish, O F, and Hartwell, W V 1951 Pantothenic acid deficiency in baby pigs Jour Anim Sci 10 80

Wilson, F M 1910 Enteritis in swine Jour Amer Vet Med Assn 96 141

Wintrobe M M 1939 Nutritive requirements of young pigs Amer Jour Phys 126 375

———, Alcayaga, R Humphreys, S, and Follis, R H, Jr 1943a Electrocardiographic changes associated with thiamine deficiency in pigs Johns Hopkins Hosp Bull 73 169

———, Buschke, W, Follis R H, Jr, and Humphreys, S 1944 Riboflavin deficiency in swine Johns Hopkins Hosp Bull 75 102

———, Follis R H Jr, Alcayaga, R, Paulson, M, and Humphreys, S 1943b Pantothenic acid deficiency in swine with particular reference to the effects in growth and on the alimentary tract Johns Hopkins Hosp Bull 73 313

———, Miller, M H, Stein, H J, Alcayaga, R, Humphreys, S, Suksta, A, and Cartwright, G E. 1943c. Pyridoxine deficiency in swine Johns Hopkins Hosp Bull 72 1

———, Miller, M H Follis R H, Jr Stein, H J, Mushatt, C, and Humphreys, S 1942b Sensory neuron degeneration in pigs Jour Nutrit 24 345

———, Stein, H J, Miller, M H, Follis, R H, Jr and Humphreys, S 1945 Nicotinic acid and the level of protein intake in the nutrition of the pig Jour Nutrit 30 395

———, Miller, M H, Follis, R H, Jr, Najjar, V, and Humphreys, S 1942a A study of thiamine deficiency in swine Johns Hopkins Hosp Bull 71 141

Witts, L J 1956 Recent work on B vitamins in the blood and gastrointestinal tract, especially in relation to human diseases Brit Med Bull 12 14

Witz, W M, and Beeson, W M 1951 The physiological effects of a fat deficient diet on the pig Jour Anim Sci 10 112

Wolbach, S B, and Bessey, O A 1942 Tissue changes in vitamin deficiencies Physiol Rev 22 233

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CHAPTER 50

Parakeratosis

Parakeratosis is a disease of the skin More especially it is a disease of the epidermal layer of the skin Parakeratosis is a sub acute chronic noninflammatory and self limiting disease that usually terminates in complete recovery Early in its clinical course small papules develop in the skin of the ventral abdominal wall and medial surface of the thighs Later it is marked by hard dry crusted proliferations on the distal parts of the legs tail ears face shoulders thighs and sides of the body The disease occurs most often in pigs be tween the ages of 7 and 20 weeks It is a metabolic disease in which the essential fatty acids appear to have an etiological role of fundamental importance

The term parakeratosis was suggested as a name for this disease by Kernkamp and Ferrin (1953) The marked accumulation of the desquamating cornified layer of the epidermis in which many of the nuclei are retained and in which there are masses of keratohyalin granules is the most char acteristic tissue alteration These changes constitute a basis for the use of the term parakeratosis Kernkamp and Ferrin have observed this disease in Minnesota each year since 1942 and have looked upon it as a disease entity for most of that time

More and more since 1940 veterinarians nutritionists husbandmen and swine pro ducers have been cognizant of the presence of a disease of the skin of feeder pigs in particular that was not typical and char acteristic of diseases more familiar to them

During this period reports from several agricultural experiment stations contained references to the occurrence of a disease of the skin of swine which developed in connection with experiments These ex periments were designed to study the qualitative and quantitative values of ra tions compounded from various dietary substances The descriptions and/or illus trations of the skin disorder leave little doubt that it was parakeratosis Hogan and Johnson (1941) called attention to the occurrence of a heavy brown exudate on the skin of some of the pigs they were feeding Keith et al (1942) described their cases as a dermatitis and suggested the dis ease be known as epidermidosis because only the epidermal layer of the skin was involved Krider et al (1944) used the term elephant hide to denote the thick harsh and dry nature of the skin Accord ing to Cunha et al (1944) the lesion rep resented an exudate on the skin surface and Ross et al (1944) referred to it as a dermatosis. It was called scabby skin by Robinson (1948) and scaly skin by Lehrer and Wiese (1952) Witz and Beeson (1951) referred to the disease they pro duced in pigs by means of fat deficient diets as scaly dandruff

Mohler (1911) described a disease of the skin of swine which he called elepf in tiasis papillomatosi The disease involved all or parts of the body surface The skin is said to have been much thicker and rougher than normal and to have formed

deep wrinkles on the head, neck, and sides of the body Sebaceous material and debris lodged in these creases Mohler claimed, that the disease was not of a contagious nature It is very probable that the pigs were affected with the same disease as is described here

Parakeratosis is widespread in North America Reports of its occurrence have been received by the writer from all sections of the United States and from many parts of Canada Stevenson and Earle (1956) reported the occurrence of this disease in England Denmark, and Sweden, and Perpere and Placidi (1956) observed it in France

Generally speaking, parakeratosis is more prevalent in winter and early spring in regions where the atmospheric temperature reaches zero and subzero values at these times of the year It has been the observation of persons familiar with the disease that it occurs much less frequently among pigs whose diets include pasture grasses

ETIOLOGY

The etiology of parakeratosis appears to be involved in a metabolic relationship of calcium and zinc and a relative deficiency of essential fatty acids The relation of high calcium rations to its occurrence as well as the beneficial therapeutic effects that have followed the supplemental feeding of zinc salts was reported by Brinegar (1955), Tucker and Salmon (1955) Hockstra (1955), Luecke *et al* (1956), and Stevenson and Earle (1956) Subsequently, Hanson and Sorensen (1957) carried on studies which suggest that parakeratosis is closely related to an essential fatty acid deficiency The deficiency is a relative deficiency which, according to these workers, arises as a result of an increased need for these acids by the rapidly growing pigs They point out that several different factors or circumstances contribute to the over all etiological aspects of this disease It should be noted, they say, that parallel with the occurrence and recognition of the disease considerable advances were being made in increasing the growth rates of pigs through the development of new breeds and strains

At the same time, rapid advancements in the area of nutrition and dietary supplements was taking place The addition to the diet of vitamin B_{12} and of some antibiotics resulted in significant growth stimulation in many instances

Hanson and Sorensen make a point of the fact that swine rations in general are low in lipids and especially lipids of the unsaturated fatty acids While it appears, with few exceptions, that pigs can synthesize all the lipid constituents required by the body from non lipid material — carbohydrates and proteins — it also appears that during periods of extremely rapid growth, the biosynthesis of the unsaturated fatty acids does not keep up with their need Parakeratosis was readily produced in the pigs that had the ability for rapid growth and that were receiving a growth promoting ration At the same time it was prevented in another group of comparable pigs receiving the same ration except that it was fortified with a higher proportion of fatty acids

The interrelationships and mechanisms of the action between the minerals and fatty acids should be considered in the over all picture of the etiology, but as yet these have not been adequately investigated and they require further study

CLINICAL SIGNS

The symptom which characterizes parakeratosis is the development of keratinous crusts on the surface of the skin They represent the final stage of development of the lesion (Figs 50 1 and 50 2)

In the very early phases of the disease the lesion is a more or less circumscribed erythematous area in the skin This is followed by a circumscribed elevated area, 3–5 mm in diameter which is soon overlaid with scales These occur most commonly on the ventral and ventrolateral surface of the abdomen and medial surface of the thigh The macule and papule stages are of relatively short duration and pass quickly to stages which more definitely characterize the disease The pastern, fetlock, knee, and hock regions of the legs are areas where the keratinous crusts

usually occur early and are the first clinical evidence of the disease that can be noted without catching and restraining the pig for examination at close range In some cases crusts occur on the tail, ear, shoulder hip, or thigh at an early period In still others, keratinous lesions occur early in two or more of these sites or regions simul taneously The lesion fails to extend much beyond its original locus in some pigs but in most cases it spreads until it affects a large area of the body The entire body surface is involved in some cases

It is not uncommon to find a symmetri cal distribution of the lesions For example if the hock and cannon regions of the left leg are involved the hock and cannon regions of the right leg will be involved Likewise if an area of the abdominal wall on the right side is affected a similar area and location on the left side will be af fected

The crusts fissure and crack to form hummocks of keratinous epidermal tissues of various size and shape They sometimes measure 5–7 mm in thickness and as a

FIG 50 1 — Keratinous crusts on skin of pasterns fetlocks hocks, thigh, and w thers
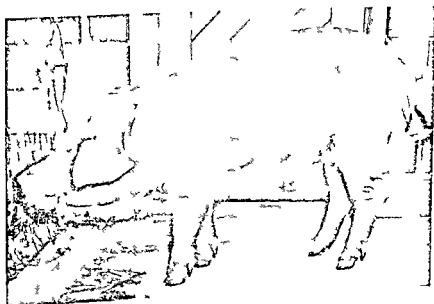
FIG 50 2 — Keratinous crusts on sk n of practic ally the entire body
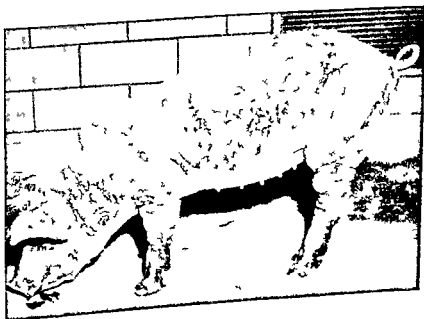
rule, are not firmly attached to the underlying *cutaneous structures*. The free surface of the hummocks is dry, horny, granular like and rough. The hairs are often entangled and matted in the proliferated tissue and, as a rule, they do not break off or pull out easily. The crusts can often be crumpled and removed from the skin by slight rubbing with the fingers and thumb. They are not scales or flakes and do not form branny layers. They are not greasy except where the exudate of some infective process acting on some of the underlying cutaneous structures creates a moistened tissue aggregate.

The fissures or cracks often contain a moist, somewhat sticky, brownish black substance—an admixture of sebum, particles of soil, litter, and other debris. In some cases, the derma at the bottom of a fissure becomes infected and produces a very localized inflammatory reaction. This is a secondary change which complicates the existing disease. The lesions of parakeratosis *per se* do not cause the patient much discomfort or distress. There is a minimum of rubbing and scratching.

Usually, the feed intake of pigs that are rather severely affected with the disease is markedly reduced. In line with this the rate of gain in weight is impaired or there may be a weight loss. On the other hand, in the less severe or extensive cases a good appetite is retained and the rate of gain may continue in a satisfactory manner. The feces of some pigs affected with parakeratosis may be soft and of a consistency that is often referred to as a scour.

The clinical course of the disease varies with the extent and severity of the involvement and with conditions and circumstances that pertain to the diet. Complete recovery of some mild and even moderately severe cases has occurred in from 10 to 14 days without any change in the diet or management. By the same token severe cases have cleared up in 30 to 45 days. Loss by death from this disease is an exceptionally rare occurrence. Economic loss due to a retardation of growth is directly related to the severity of the disease, the cost of

feedstuffs, and the time that it takes to effect a satisfactory recovery.

PATHOLOGICAL CHANGES

Since the gross pathological changes that occur in this disease are confined to the skin, they have been discussed in connection with a description of the clinical signs. In a full-blown and typical case of parakeratosis the histopathology of the skin shows that the disturbance involves the epidermis in particular (Fig. 50.4). Excepting the *stratum lucidum*, there is a marked increase in all of the epidermal elements. The crusted masses on the outer surface of the skin are composed of a large accumulation of the cornified epithelium —*stratum corneum*—that are sometimes arranged in definite layers but more often in irregular masses and/or conglomerations of layered and irregular masses. A characteristic feature is the presence in this outer layer of epithelium of many nuclei and collections of keratohyalin granules. Another important alteration is an increase in the number and length of the rete pegs that dip down into the derma portion of the skin.

DIFFERENTIAL DIAGNOSIS

It is important to differentiate between parakeratosis and other disorders of the skin with which it might be confused. Parakeratosis is most often mistaken for sarcoptic mange. The early lesions of mange are reddened papules or sometimes vesicles that are covered at first by dry, branlike scales and later by a dark-brown crust. In severe cases the skin becomes thickened and deploys in large folds. The lesions usually occur around the ears, medial surface of the thigh, or sides of the body. From here they spread to cover large areas of the body. The activities of the mange mites produce an intense irritation of the skin and cause the pigs to rub or scratch themselves vigorously against any fixed objects they can contact, such as posts, feeding and watering utensils, and corners of buildings. The condition may progress to the point of causing cachexia and death.

If the disease is mange, an examination of suitable skin scrapings will reveal the presence of the parasite Sarcoptic mange and parakeratosis may occur simultaneously

'Greasy pig disease,' or exudative epidermitis, is also confused with parakeratosis This disease, however, usually occurs in younger pigs than are affected by parakeratosis and is characterized by a more greasy exudative surface The rancid disagreeable odor of exudative epidermitis is not common in parakeratosis

TREATMENT

A first and early matter for consideration in connection with the therapeutic management of pigs affected with parakeratosis is to ascertain the composition of the diet they are presently receiving and

FIG 50 3 — Normal skin from region of thigh The absence of nuclei in the cornified layer (stratum corneum) is conspicuous

FIG 50 4 — Parakeratotic skin from region of thigh The cornified layer is irregularly thickened and many nuclei, much keratohyalin material, and debris are within it Note also that the long rete pegs of the Malpighian layer dip into the dermis
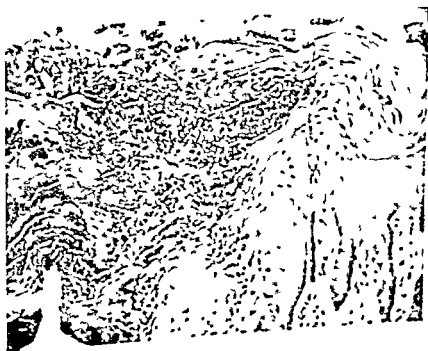
the length of time they have received it prior to the onset of the disease Special emphasis should be given to the proportion of calcium in the diet. If the calcuhted level of calcium is 1 per cent or more it is imperative that the ration be changed so that the level of calcium is between 0 60 and 0 75 per cent There is considerable evidence to show that diets high in calcium aggravate this disease Rations that will supply approximately 12 lb of calcium per ton of feed will furnish it at the rate of about 0 60 per cent It is advisable

also to supplement the ration with a zinc salt at the rate of 0 02 per cent This requires that 0 4 lb of zinc carbonate or zinc sulfate be added to a ton of feed Where prompt and speedy curative results are desired, it would be advisable to add to the ration a soybean oil that contains the essential fatty acids A refined product containing 54 per cent of linoleic acid produced some very satisfactory results when mixed into the ration at the rate of 20 per cent by weight It should be made up fresh every day to avoid rancidity

REFERENCES

BRINEGAR M J 1955 Personal communication

CUNHA T J Ross O B PHILLIPS P H AND BOHSTEDT, G 1944 Further observations on the dietary insufficiency of a corn soybean ration for reproduction of swine Jour Anim Sci 3 415

HANSON, L J AND SORENSEN D K 1957 Unpublished data

HOLKSTRA W G 1955 Parakeratosis in swine Vet Sci News Univ Wis 9 1

HOGAN A S AND JOHNSON S R 1941 Supplementary value of various feedstuffs in brood sow rations Mo Agr Exp Sta Res Bull 332

KEITH T B MILLER R C THORP W T S AND MCCARTY M A 1942 Nutritional deficiencies of a concentrate mixture composed of corn tankage soybean oil meal and alfalfa meal for growing pigs Jour Anim Sci 1 120

KERNKAMP H C H AND FERRIN E F 1953 Parakeratosis in swine Jour Amer Vet Med Assn 123 417

KRIDER J L FAIRBANKS B W AND CARROLL, W E 1944 Distillers by products in swine rations Jour Anim Sci 3 107

LEHRER W P AND WIESE A C 1952 Riboflavin deficiency in baby pigs Jour Anim Sci 11 211

LUECKE R W HOEFER J A BRAMMELL, W S AND THORP F JR 1956 Mineral interrelationships in parakeratosis in swine Jour Anim Sci 15 317

MOHLER J R 1911 Elephantiasis papillomatosa U S D A Bur Anim Ind 28th Ann Rept p 62

TERRIER I AND FLACHT L 1956 "Parakeratose syndrome de desequilibre alimentaire chez le porc. Rec. méd Vet 132 913

ROBINSON W L 1948 Solvent extracted cottonseed meal as a protein concentrate for pigs in dry lot Jour Anim Sci 7 531

Ross O B PHILLIPS P H BOHSTEDT G AND CUNHA T J 1944 Congenital malformations syndactylism talipes and paralysis agitans of nutritional origin in swine Jour Anim Sci 3 406

STEVENSON J W AND EARLE I P 1956 Studies on parakeratosis in swine Jour Anim Sci 15 1054

TUCKER H F AND SALMON W D 1955 Parakeratosis or zinc deficiency disease in the pig Proc Soc Exper Biol and Med 88 613

WITZ W M, AND BEESON W M 1951 The physiological effects of a fat-deficient diet on the pig Jour Anim Sci 10 112

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Feeds and Feeding

The major function of swine on American farms is the conversion of products of the farm into high value items for human consumption The swine producer expects to realize a profit from the process In most manufacturing enterprises, the net return to the manufacturer is determined by the difference between the cost of production and the selling price This is true of the business of swine raising Unfortunately, too many swine producers show more interest in the price they expect to get for their market animals than they do in the cost of producing those hogs The producer actually has little control over the price he gets on the market, while he can exert considerable influence on the cost of production It should be noted that feed costs make up about 79 per cent of the total cost of producing hogs for slaughter This fact makes it obvious that, since the cost of the feed invested in every 100 lb of live hog marketed represents such a large proportion of the total investment, it is extremely important that the feed cost be kept at a minimum This can be done by feeding rations which are

1 Balanced nutritionally for the particular class of swine being fed
2 Compounded from ingredients which are palatable and safe to feed
3 Compounded from ingredients which supply their particular nutrients most economically
4 Composed of ingredients which will not produce an inferior product

Swine are quite efficient in their conversion of feed into body gain, being excelled only by broiler chickens Growing pigs require considerably less feed per pound of gain than do calves or lambs It is not unreasonable to expect well bred pigs fed a balanced ration to make a pound of gain from less than three pounds of feed, on the average, during the period between weaning and the attainment of 200 lb live weight

BALANCED RATIONS

A nutritionally balanced ration is one which supplies all the nutrients required by an animal in such proportions and amounts as to provide for maximum production and with a minimum of nutrient wastage Modern knowledge of the nutrient requirements of swine together with the available information concerning the nutrient content of feed ingredients makes it possible to formulate rations which give excellent results Even today, however, the picture is not complete The requirements for all identified nutrients for all classes of swine are not known, and there are apparently some required nutritional factors which have not been identified but which are contained in natural feedstuffs such as pasture, alfalfa meal, and others

The extreme importance of proper ration formulations for the efficient utilization of feed by swine has been demonstrated at the University of Minnesota by Hanson (1954) He fed three types of

rations in dry lot to groups of weanling pigs which averaged 51 lb at the beginning of the trial The three types of rations were typical of those fed in 1910 1930 and 1953, respectively The 1910 ration was composed of 97 per cent ground yellow corn and 3 per cent minerals and included vitamins A and D although they were not known in 1910 The ration fed in 1930 was composed of ground yellow corn dry rendered tankage, minerals and vitamins A and D The ration typical of 1953 included ground yellow corn, solvent process soybean oil meal, dry rendered tankage, linseed meal, alfalfa meal, minerals vitamin D_2 vitamin B_{12} pantothenic acid niacin, choline folic acid, and an antibiotic feed supplement The greatest contrast in efficiency of feed utilization was between the 1910 and the 1953 ration The pigs fed the former required 12 1 bu of corn and 21 lb of minerals per 100 lb of gain, while those fed the latter required only 5 2 bu of corn and 52 lb of supplemental feeds to produce 100 lb of gain The pigs fed the more modern ration also gained faster

NUTRIENT REQUIREMENTS FOR SWINE

Table 51 1 presents the nutrient requirements for swine as they were summarized by the Committee on Swine Nutrition of the National Research Council (Beeson *et al*, 1953) Requirements are presented for the nutrients for which there are reasonably reliable quantitative data given in the literature

Quantitative data for some of the nutrients required by swine are not known This is particularly true for brood sows performing the functions of gestation and lactation As new data become available modification and extension of the present requirements will be possible

Nutrients which are labile in mixed feeds probably should be provided at levels somewhat higher than are given in the table if the feed is kept in storage The requirements for swine of the essential amino acids and the trace minerals will be discussed later

SOME IMPORTANT FEED INGREDIENTS

Swine have digestive tracts which are limited in capacity as compared to cattle, sheep, and horses Because of this it is impossible for them to make efficient use of relatively large amounts of roughage This means that most classes of swine should be fed rations which are concentrated — rations which are rich in digestible nutrients and low in fiber Such rations are built around the cereal grains, with corn being the one most widely used

The cereal grains are high in their content of readily available energy but they have serious nutrient deficiencies In order to formulate a balanced ration using grain as the major ingredient, it is necessary to include a combination of feedstuffs to supply the nutrients in which the grain is deficient

Corn grain is an excellent source of energy The fiber content is quite low even when compared with the other grains The protein content of corn is low and the quality of the protein is poor because of the low levels of the two essential amino acids, lysine and tryptophan Corn is quite deficient in calcium and is only a fair source of phosphorus Corn is also deficient in its content of salt, vitamin D riboflavin, pantothenic acid, choline, and vitamin B_1. Yellow corn is a fair source of carotene

Oats are considerably higher in fiber than corn and because of this are not the equivalent of corn in rations for younger pigs when used as the major grain However when the proportion of oats is limited to not more than 25 per cent of the complete ration and when full advantage is taken of their higher protein content oats are equivalent to corn pound for pound Oats are quite low in calcium and only fair in phosphorus Oats contain little if any carotene or vitamin D and are a poor source of riboflavin This grain is a fairly good source of both pantothenic acid and choline supplying about twice the amount of each that is contained in corn Oats are very low in vitamin B_{12}

TABLE 51 1

NUTRIENT REQUIREMENTS FOR SWINE IN PERCENTAGE OR AMOUNT PER POUND OF TOTAL RATION

	DESCRIPTION OF PIGS									
	Market Stock						Breeding Stock			
							Pregnant females and Breeding Boars		Lactating females	
							Young Stock	Adults	Gilts	Adults
Live weight, lb	25	50	100	150	200	250	300	500	350	450
Expected daily gain, lb	0.8	1.2	1.6	1.8	1.8	1.8	0.75	0.5	0.75	
Total digestible nutrients, lb	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75
Crude protein, %	18.0	16.0	14.0	13.0	12.0	12.0	15.0	14.0	15.0	14.0
Inorganic nutrients										
Calcium, %	0.8	0.65	0.65	0.55	0.55	0.55	0.6	0.6	0.6	0.6
Phosphorus, %	0.6	0.45	0.45	0.33	0.33	0.33	0.4	0.4	0.4	0.4
Salt (NaCl), %	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Vitamins										
Carotene, mg	0.25						2.5	2.5	2.5	2.5
Vitamin D, I U	90	90	90	90	90	90	90	90	90	90
Thiamine, mg	0.1	0.1	0.1	0.1	0.1	0.1	0.5	0.5	0.5	0.5
Riboflavin, mg	0.2						1.2	1.2	1.2	1.2
Niacin, mg	8.0									
Pantothenic acid, mg	6.0									
Pyridoxine, mg	0.6									
Choline, mg	400.0									
Vitamin B_{12}, μg	7.0		5.0				4	4	4	4

rations in dry lot to groups of weanling pigs which averaged 51 lb at the beginning of the trial The three types of rations were typical of those fed in 1910, 1930 and 1953, respectively The 1910 ration was composed of 97 per cent ground yellow corn and 3 per cent minerals and included vitamins A and D although they were not known in 1910 The ration fed in 1930 was composed of ground yellow corn dry rendered tankage, minerals, and vitamins A and D The ration typical of 1953 included ground yellow corn, solvent process soybean oil meal, dry rendered tankage, linseed meal, alfalfa meal, minerals, vitamin D_2, vitamin B_{12} pantothenic acid, niacin, choline folic acid, and an antibiotic feed supplement The greatest contrast in efficiency of feed utilization was between the 1910 and the 1953 ration The pigs fed the former required 12 1 bu of corn and 21 lb of minerals per 100 lb of gain, while those fed the latter required only 5 2 bu of corn and 52 lb of supplemental feeds to produce 100 lb of gain The pigs fed the more modern ration also gained faster

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Nutrients which are labile in mixed feeds probably should be provided at levels somewhat higher than are given in the table if the feed is kept in storage The requirements for swine of the essential amino acids and the trace minerals will be discussed later

SOME IMPORTANT FEED INGREDIENTS

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The cereal grains are high in their content of readily available energy but they have serious nutrient deficiencies In order to formulate a balanced ration using grain as the major ingredient, it is necessary to include a combination of feedstuffs to supply the nutrients in which the grain is deficient

Corn grain is an excellent source of energy The fiber content is quite low even when compared with the other grains The protein content of corn is low and the quality of the protein is poor because of the low levels of the two essential amino acids, lysine and tryptophan Corn is quite deficient in calcium and is only a fair source of phosphorus Corn is also deficient in its content of salt, vitamin D riboflavin pantothenic acid, choline, and vitamin B_{12} Yellow corn is a fair source of carotene

Oats are considerably higher in fiber than corn and because of this are not the equivalent of corn in rations for younger pigs when used as the major grain However, when the proportion of oats is limited to not more than 25 per cent of the complete ration and when full advantage is taken of their higher protein content oats are equivalent to corn pound for pound Oats are quite low in calcium and only fair in phosphorus Oats contain little if any carotene or vitamin D and are a poor source of riboflavin This grain is a fairly good source of both pantothenic acid and choline, supplying about twice the amount of each that is contained in corn Oats are very low in vitamin B_{12}

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							Pregnant Females and Breeding Boars		Lactating Females	
							Young Stock	Adults	Gilts	Adults
Live weight, lb	25	50	100	150	200	250	300	500	350	450
Expected daily gain, lb	0 8	1	1 6	1 8	1 8	1 8	0 75	0 5	0 75	0 75
Total digestible nutrients, lb	18 0	16 0	14 0	13 0	12 0	12 0	15 0	14 0	15 0	14 0
Crude protein, %										
Inorganic nutrients										
Calcium, %	0 8	0 65	0 65	0 55	0 55	0 55	0 6	0 6	0 6	0 6
Phosphorus, %	0 5	0 45	0 45	0 33	0 33	0 33	0 5	0 4	0 4	0 4
Salt (NaCl), %	0 5	0 5	0 5	0 5	0 5	0 5	0 5	0 5	0 5	0 5
Vitamins										
Carotene, mg	0 25	0 31	0 38	0 44	0 53	0 60	2 5	2 5	2 5	2 5
Vitamin D, I	90	90	90	90	90	90	90	90	90	90
Thiamine, mg	2	2	2	1	1	1	2	2	2	2
Riboflavin, mg	1	1	1	1	1	1	1	1	1	1
Niacin, mg	8 0	5 0	5 0	5 0	5 0	5 0	5 0	5 0	5 0	5 0
Pantothenic acid, mg	6 0	2 0	2 0	0	0	0	1	1	1	1
Pyridoxine, mg	6 0	5 0	5 0	5	5	5	5	5	5	5
Choline, mg	400 0	5 0	4	4	4	4	5	4	4	5
Vitamin B12, mg	7 0									

In sections of the country where the growing season is too short for corn to mature barley is often used as the major grain for swine feeding Barley is almost half again as rich in protein as corn, but its protein is not of good quality Barley is much higher in fiber than corn and therefore is not equal to corn in feeding value even though it is higher in protein Barley is deficient in calcium but is a fair source of phosphorus The grain is very low in carotene, vitamin D, and vitamin B_{12}, and is poor in riboflavin content. The pantothenic acid level of barley is not much higher than that of corn, but barley is a good source of choline Growing and fattening pigs self fed free choice on barley sometimes tire of it after several months

Wheat is an excellent grain for swine feeding, but wheat of milling grade is usually too expensive to be used economically as a feed Wheat is worth slightly more per unit weight than corn It is much richer in protein than corn and its protein is of somewhat better quality As with barley and oats, wheat is a very poor source of carotene, vitamin D, and vitamin B_{12} Wheat is quite deficient in calcium but is a fair source of phosphorus This grain is low in riboflavin but is a good source of pantothenic acid and choline

The high protein feed ingredients useful in swine feeding are usually classified according to origin those of plant origin and those made from animal materials including fish by products and milk by products The most commonly used plant protein supplements are soybean oil meal, cottonseed meal, and linseed meal The solvent process of extracting oil from these oil bearing seeds is the method most widely used today

Soybean oil meal is the most popular of the three because it is rich in protein of excellent quality and is well liked by swine However, soybean oil meal has certain nutrient deficiencies which must be noted when it is used in a ration with grain The calcium level is low It is quite deficient in salt and may be somewhat deficient in the essential amino acid,

methionine Soybean oil meal is fairly rich in phosphorus This meal supplies little if any carotene, vitamin D, and vitamin B_{12} It is a fair source of riboflavin, a good source of pantothenic acid, and is rich in choline

Soybean oil meal must be properly cooked if it is to have its maximum feeding value The meal is so palatable to swine that they often eat more than is necessary to balance the ration for protein when it is self fed free choice with grain Soybean oil meal is used as a supplement to grain to raise the level of protein, to improve the quality of the protein in the ration, and to increase the levels of pantothenic acid and choline in the ration A dehulled soybean oil meal is available which is higher in protein and lower in fiber than the standard solvent process meal

Cottonseed meal often contains toxic amounts of a poison called gossypol However, low gossypol cottonseed meals are available which can be fed with no danger of poisoning The protein of cottonseed meal is of poor quality and it should not be used as the only source of supplementary protein in swine rations It is satisfactory, however, in supplemental mixtures which contain animal protein such as meat scrap Cottonseed meal is quite deficient in carotene, vitamin D, and vitamin B_{12}, as well as in calcium and salt This meal is quite rich in phosphorus It is a fair source of riboflavin, a good source of pantothenic acid, and an excellent source of choline

Linseed meal is quite popular with some swine feeders for certain rations because of its slightly laxative effect and because it tends to increase the glossiness of the hair coat Linseed meal provides protein of only fair quality and should be fed in combination with protein supplements such as meat scrap or fish meal Linseed meal is fair in calcium content and has a relatively high phosphorus level It supplies little if any carotene, vitamin D, and vitamin B_{12} It also is lacking in salt The riboflavin level of this meal is fair as is

the level of pantothenic acid It is not as rich in choline as soybean oil meal or cot tonseed meal

Cottonseed meal and linseed meal are usually included in a ration to increase the protein level and to improve the quality of the protein mixture somewhat Since these meals are richer than the grains in riboflavin, pantothenic acid and choline, their inclusion in the mixture raises the level of these factors in the ra tion

Whole milk is usually too expensive to use as a swine feed All milk by products supply protein of excellent quality and some of them are fairly rich in protein Liquid skim milk and buttermilk are about equal in feeding value They can be fed economically when the cost of the skim milk or buttermilk per 100 lb is roughly equal to or less than the cost of ¼ bu of corn or 12 lb of tankage These products have their greatest value in swine rations when fed with grain in amounts which just balance the ration for protein When corn is self fed free choice to grow ing and fattening pigs in dry lot one gallon of skim milk or buttermilk per head per day will balance the protein in the ration Some source of carotene and vitamin D may be needed because these milk by products are rather low in these vitamins

Fish meal is an excellent protein sup plement for use in swine rations Usually it is too expensive to be fed as the only source of supplementary protein Fish meal is occasionally included in small amounts in pig prestarters and pig start ers It is quite high in protein content and the protein is of excellent quality Fish meal is a very good source of calcium phosphorus, and salt but is not a reliable source of vitamin A or vitamin D The meal is fair in pantothenic acid content, is a good source of riboflavin, and is rich in vitamin B_{12} There are several kinds of fish meal according to the kind of fish in volved

Wet rendered or digester tankage has been used by swine feeders for many years It is a packing house by product which is

quite high in protein level The quality of the protein in this material is only fair but it is more effective than cottonseed meal or linseed meal in correcting the essential amino acid deficiencies of the grains It is an excellent source of calcium phosphorus and salt but it supplies little if any carotene or vitamin D Tankage is relatively rich in vitamin B_{12} but it con tains rather low levels of riboflavin and pantothenic acid It is not as rich as the oil meals in choline

Meat and bone scrap of 50 per cent protein grade is a dry rendered packing house by product which is superior to digester tankage in protein quality It is richer in calcium and phosphorus than digester tankage Meat and bone scrap contains no appreciable amount of caro tene or vitamin D and is only fair in its riboflavin and pantothenic acid content It is a good source of vitamin B_{12} and is fairly high in choline Meat and bone scrap can be used to replace tankage pound for pound with no loss in the per formance of the swine being fed

When swine are being fed on good actively growing pasture, the pasture that they eat insures them against any vitamin deficiency except possibly vitamin B_{12} Figure 51 1 shows an example of a good stand of ladino clover pasture which is excellent for swine When pasture is not available, the inclusion of 10 per cent or more of high quality legume hay or meal in dry lot rations is almost as effective in preventing vitamin deficiencies as the pas ture Thus high quality legume hay or meal serves as a vitamin supplement Al falfa hay or meal is the most commonly used legume hay or meal It is an excellent source of carotene and if sun cured, a satisfactory source of vitamin D Alfalfa hay is a good source of riboflavin and pantothenic acid and is fair in choline content The hay supplies very little vita min B_{12} High quality alfalfa hay supplies considerable protein which is of fairly good quality The hay is relatively rich in calcium but is not a good source of phosphorus

FIG 51.1 — Ladino clover makes good pasture for swine. (Photo courtesy E. A. Hollowell, U S D A.)

Distillers dried corn solubles is used as a supplementary source of riboflavin, pantothenic acid, and choline, being a good source of these vitamins. Fermentation solubles is also used to supply these vitamins since it is especially rich in them. Condensed fish solubles is used for the same purpose.

ENERGY REQUIREMENTS

Energy is required for body maintenance and the productive functions which include growth and fattening, reproduction, and lactation. Table 51.1 indicates that all classes of swine being fed for maximum production should be fed rations containing 75 per cent total digestible nutrients. This high level of digestible energy can be assured by using grains such as corn or barley as the major ingredient in the ration and by limiting the fiber content by, in turn, limiting the amounts of fibrous ingredients such as oats and hay. Carroll and Krider (1953) suggest the following maximum levels of crude fiber in rations for various classes of swine:

1. Growing and fattening pigs, 6 to 8 per cent
2. Bred sows, 10 to 12 per cent.

Adjustment of the crude fiber content of the self-fed ration can be used to regulate the energy intake of bred sows and gilts and other classes of swine for which, because of the possibility of excessive fat-ness, maximum energy intake is not desired.

PROTEIN REQUIREMENTS

The quantitative requirements for the different classes of swine for total protein, expressed as a per cent of the total ration, are given in Table 51.1. These requirements are for dry-lot feeding. When swine are grazing on good pasture, these figures may be reduced somewhat because of the protein supplied by the pasture the animal consumes.

Dietary protein supplies amino acids used by the animal in replacing body proteins lost through endogenous catabolism and in the formation of new protein tissue produced in growth, reproduction, and lactation.

The grains, even though they are rich in energy, do not supply a level of protein high enough to meet the requirements of most classes of swine. This makes it necessary to include in the diet one or more of the high protein supplements which are suitable for swine feeding. Some of these have been mentioned.

There are several methods used in adding protein supplements to the swine ration. The proper amount of supplement can be mixed with the grain and other ingredients of the ration and the mixture either hand-fed or self-fed. Another method is to self-feed free-choice the grain in

one compartment of a self feeder and the protein supplement in another compartment This is possible because growing and fattening pigs have the ability, to a certain extent, to balance the protein level of the ration for themselves when they are self fed free choice grain and protein supplement However, some protein supplements have such palatability that the pigs eat too much or too little of them and the protein intake level is incorrect

A third method of feeding a protein supplement is to hand feed the correct amount each day to pigs being self fed grain A fourth method is to hand feed the supplement to swine being hand fed grain such as ear corn

An essential amino acid is one which cannot be synthesized by the animal at a rate which will provide for normal growth Protein quality refers to the proportions and amounts of essential amino acids present in a protein or a mixture of proteins The quality of the protein in a swine ration is very important This is particularly true for rations fed during gestation, lactation, and during the period of growth from weaning to a live weight of about 75 lb During these periods protein synthesis is proceeding at a very rapid rate Table 51 2 presents tentative requirements for the 10 essential amino acids for 25 to 70 lb pigs as suggested by the Committee on Swine Nutrition of the National Research Council (Beeson *et al*, 1953) This publication also gives some data on the amino acid content of some feedstuffs Informa

tion is lacking on the essential amino acid requirements for the classes of swine other than weanling pigs weighing 25 to 70 lb

The total protein requirements for swine as given in Table 51 2 represent the levels which will assure adequate amounts and proportions of essential amino acids when a variety of sources of protein are included in the diet It has been demonstrated by Mertz *et al* (1952) that a considerably lower level of total protein (N x 6 25) in a purified diet can produce good growth in weanling pigs when 7 4 per cent of protein was supplied by specific amino acids and 3 9 per cent of protein equivalent was supplied by diammonium citrate This total of 11 3 per cent is considerably lower than the 16 per cent recommended in Table 51 1 The work of Mertz *et al* makes possible at least two general observations

1. The weanling pig can probably synthesize nonessential amino acids from non protein nitrogen

2. The scoop shovel methods of providing essential amino acid balance in swine rations used today, i e, including a variety of protein sources, are quite wasteful of expensive high protein ingredients

More research in this area must be conducted before it will be advisable and economically feasible to formulate swine rations on the basis of synthetically produced essential amino acids and either natural protein carriers or sources of non protein nitrogen However, it is probable

TABLE 51 2
ESSENTIAL AMINO ACID REQUIREMENTS FOR WEANLING PIGS
(Live weight—25–70 lb)

Amino Acid	Percentage of Total Diet	Amino Acid	Percentage of Total Diet
L-Arginine*	0 20	DL-Methionine †	0 60
L-Histidine*	0 40	DL-Phenylalanine	0 46
L-Isoleucine	0 70	L-Threonine	0 40
L-Leucine*	0 80	DL-Tryptophan	0 20
L-Lysine	1 00	L-Valine	0 40

* This level is adequate but minimum requirement has not been established
† Cystine can replace one-half (0 3%) of the methionine requirement

FIG 51 1 — Ladino clover makes good pasture for swine (Photo courtesy E A Hollowell USDA)

Distillers dried corn solubles is used as a supplementary source of riboflavin, pantothenic acid and choline being a good source of these vitamins Fermentation solubles is also used to supply these vitamins since it is especially rich in them Condensed fish solubles is used for the same purpose

ENERGY REQUIREMENTS

Energy is required for body maintenance and the productive functions which include growth and fattening, reproduction and lactation Table 51 1 indicates that all classes of swine being fed for maximum production should be fed rations containing 75 per cent total digestible nutrients This high level of digestible energy can be assured by using grains such as corn or barley as the major ingredient in the ration and by limiting the fiber content by in turn limiting the amounts of fibrous ingredients such as oats and hay Carroll and Krider (1 53) suggest the following maximum levels of crude fiber in rations for various classes of swine

1 Growing and fattening pigs 0 to 8 per cent

ness maximum energy intake is not desired

PROTEIN REQUIREMENTS

The quantitative requirements for the different classes of swine for total protein expressed as a per cent of the total ration are given in Table 51 1 These requirements are for dry lot feeding When swine are grazing on good pasture, these figures may be reduced somewhat because of the protein supplied by the pasture the animal consumes

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one compartment of a self feeder and the protein supplement in another compartment This is possible because growing and fattening pigs have the ability, to a certain extent, to balance the protein level of the ration for themselves when they are self fed free choice grain and protein supplement However, some protein supplements have such palatability that the pigs eat too much or too little of them and the protein intake level is incorrect

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More research in this area must be conducted before it will be advisable and economically feasible to formulate swine rations on the basis of synthetically produced essential amino acids and either natural protein carriers or sources of non protein nitrogen However, it is probable

TABLE 51.2
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L-Histidine*	0 40	DL-Phenylalanine	0 46
L-Isoleucine	0 70	L-Threonine	0 40
L-Leucine*	0 80	DL-Tryptophan	0 20
L-Lysine	1 00	L-Valine	0 40

* This level is adequate but minimum requirement has not been established.
† Cystine can replace one-half (0.3%) of the methionine requirement

supplies phosphorus which is highly available as does monocalcium phosphate Steamed bone meal, Curaçao Island phosphate, and defluorinated rock phosphate are good sources Soft phosphate with colloidal clay has been tested Poor availability is reported by Chapman et al (1955) and Plumlee et al (1955), however, Gobble et al (1956) reported that soft phosphate with colloidal clay phosphorus was approximately equal in biological availability to the phosphorus of dicalcium phosphate

The element fluorine is toxic to swine if fed at more than certain levels for a sufficiently long period of time The chemical form of the fluorine affects the toxicity of the element The maximum permissible level of fluorine for complete swine rations which would assure no fluorine damage under any feeding conditions has been suggested as 0 014 per cent by the Association of American Feed Control Officials (1948) Care should be exercised to assure that rations fed to swine do not include phosphorus sources which contain fluorine to the extent that this element exceeds a level of 0 014 per cent of the total ration Raw rock phosphate and soft phosphate with colloidal clay contain about 3 5 per cent and 1 5 per cent of fluorine respectively If as much as 0 18 per cent of phosphorus from either of these sources is added to the ration, the maximum permissible level of fluorine of 0 014 per cent will be exceeded However, Gobble et al (1956) fed rations containing as much as 0 055 per cent of fluorine from soft phosphate with colloidal clay for as long as 117 days to growing and fattening pigs with no evidence of fluorine toxicosis Jordan et al (1956), on the other hand report excessive pitting and decay of molars in pigs fed for 90 days a ration containing about 0 051 per cent of fluorine from soft phosphate with colloidal clay

Phosphorus supplements are more expensive per pound than ground limestone and should be used only when additional phosphorus is needed in the ration Most phosphorus carriers supply calcium also It is important that the calcium phosphorus ratio be kept within certain limits Bohstedt (1955) has suggested as favorable a range of 1 1 to 1 5 parts of calcium to 1 part of phosphorus A ratio much greater than 1 5 1 results in decreased rate and economy of gain

Sodium and chlorine are required by swine and these elements are supplied in the form of common salt Granulated salt is preferred to the block form The plant materials fed to swine (grains and oil meals) do not contain as much salt as swine need Only the animal protein supplements contain appreciable amounts of salt and these are usually not included in swine rations at high enough levels to correct the salt deficiency of the plant material part of the rations Thus it is usually necessary to add salt to the diet The recommended level, as seen in Table 51 1, is 0 5 per cent of the total ration It can be self fed free choice The work of Vestal (1946 1947a) has demonstrated very well the value of salt in a ration composed of corn soybean oil meal, alfalfa meal limestone and steamed bone meal Vestal (1947b) also found that when half the soybean oil meal in the ration was replaced with meat and bone scrap, the pigs ate only half as much free-choice salt and that adding salt to a ration of grain and meat scrap or tankage did not improve rate or efficiency of gain

There apparently is some concern among swine feeders about salt poisoning in swine This condition is discussed in detail by Whitehair in Chapter 19 In general it is recommended that swine feeders avoid feeding rations high in salt and palatable to pigs, such as brine whey or slop It is also recommended that swine always be supplied with the proper amount of salt in their rations or that loose salt be self fed free-choice at all times. It is also good practice to provide ample trough space and fresh water in plentiful amounts for the animals.

Iron is required for the formation of hemoglobin and for the prevention of nutritional anemia in suckling pigs (see

that this eventually will occur Beeson (1956) has discussed, in an illuminating manner, the protein amino acid problem in swine feeding as it exists today

Methionine is the only essential amino acid which is cheap enough to be used economically to supplement a deficiency present in swine rations

Corn is deficient in the two essential amino acids, lysine and tryptophan In order to correct these deficiencies in a practical swine ration, it is necessary to include protein supplements which are fairly rich in lysine and tryptophan Tankage and meat and bone scrap are somewhat low in tryptophan but are fairly good sources of lysine Cottonseed meal and linseed meal are fair sources of tryptophan but are a little low in lysine A ration composed of corn, tankage, and cottonseed meal provides a protein mixture of better quality than corn and tankage or corn and cottonseed meal The essential amino acid balance of soybean oil meal, fish meal, and skim milk is good However, a ration of corn, soybean oil meal, and tankage, or one of corn, fish meal, and soybean oil meal supplies protein of better quality than rations supplemented with only fish meal or soybean oil meal The superiority of swine rations supplemented with a variety of protein sources over those with single added protein supplements was first reported by Morrison and Fargo (1922) Their work developed the 'trio' mixture which may be called the grandfather of modern protein supplemental mixtures The original trio mixture was composed of 50 parts tankage, 25 parts linseed meal, and 25 parts ground alfalfa hay This mixture was fed with corn in such amounts that the quantitative protein requirement was met. The variety of protein sources assured an improvement in protein quality In addition to this, the vitamins supplied by the hay helped to meet the vitamin requirements There have since been many variations in the original trio mixture, and modern protein supplements, which include not only a variety of protein sources and minerals and vitamin supplements as

antibiotics as well, were developed from the trio mixture

MINERAL REQUIREMENTS

Swine require dietary sources of the following so-called mineral elements calcium, phosphorus, sodium, chlorine, iron, copper, cobalt, manganese, magnesium, potassium, sulfur, iodine, and zinc There is some possibility that others, including fluorine and molybdenum, are also required

These required minerals perform a wide variety of functions in the animal body Calcium is required for bone formation acid base balance, normal reproduction, and heart beat, among other things The calcium in legume pasture and hay and the protein supplements of animal origin aid materially in meeting the pig's requirements for this element When additional calcium is needed in the ration and sufficient phosphorus is present, raw ground limestone is an economical source Ground oyster shell may also be used

Table 51 1 indicates that the calcium requirement varies from 0 8 per cent to 0 55 per cent of the entire ration Excessive calcium in the diet decreases growth rate and efficiency of feed utilization and often causes a dermatitis resembling mange in appearance Adequate vitamin D is required for the utilization of calcium and phosphorus

Phosphorus is involved in bone formation, carbohydrate metabolism, fat transfer, and cell formation Although plant materials contain phosphorus in varying amounts, some of the phosphorus is present in the form of phytin There is some evidence that growing and fattening pig do not utilize plant phosphorus or phytin phosphorus as well as that from inorganic sources (Chapman *et al*, 1955, Plumlee *et al*, 1955) Some rations commonly fed to growing and fattening swine require phosphorus supplementation In selecting a phosphorus supplement some attention should be given to the availability to the pig of the phosphorus contained in the phosphatic material Dicalcium phosphate

supplies phosphorus which is highly available as does monocalcium phosphate. Steamed bone meal, Curaçao Island phosphate, and defluorinated rock phosphate are good sources. Soft phosphate with colloidal clay has been tested. Poor availability is reported by Chapman et al. (1955) and Plumlee et al. (1955); however, Gobble et al. (1956) reported that soft phosphate with colloidal clay phosphorus was approximately equal in biological availability to the phosphorus of dicalcium phosphate.

The element fluorine is toxic to swine if fed at more than certain levels for a sufficiently long period of time. The chemical form of the fluorine affects the toxicity of the element. The maximum permissible level of fluorine for complete swine rations which would assure no fluorine damage under any feeding conditions has been suggested as 0.014 per cent by the Association of American Feed Control Officials (1948). Care should be exercised to assure that rations fed to swine do not include phosphorus sources which contain fluorine to the extent that this element exceeds a level of 0.014 per cent of the total ration. Raw rock phosphate and soft phosphate with colloidal clay contain about 3.5 per cent and 1.5 per cent of fluorine, respectively. If as much as 0.18 per cent of phosphorus from either of these sources is added to the ration, the maximum permissible level of fluorine of 0.014 per cent will be exceeded. However, Gobble et al. (1956) fed rations containing as much as 0.055 per cent of fluorine from soft phosphate with colloidal clay for as long as 117 days to growing and fattening pigs with no evidence of fluorine toxicosis. Jordan et al. (1956), on the other hand, report excessive pitting and decay of molars in pigs fed for 90 days a ration containing about 0.051 per cent of fluorine from soft phosphate with colloidal clay.

Phosphorus supplements are more expensive per pound than ground limestone and should be used only when additional phosphorus is needed in the ration. Most phosphorus carriers supply calcium also. It is important that the calcium-phosphorus ratio be kept within certain limits. Bohstedt (1955) has suggested as favorable a range of 1.1 to 1.5 parts of calcium to 1 part of phosphorus. A ratio much greater than 1.5:1 results in decreased rate and economy of gain.

Sodium and chlorine are required by swine, and these elements are supplied in the form of common salt. Granulated salt is preferred to the block form. The plant materials fed to swine (grains and oil meals) do not contain as much salt as swine need. Only the animal protein supplements contain appreciable amounts of salt, and these are usually not included in swine rations at high enough levels to correct the salt deficiency of the plant material part of the rations. Thus it is usually necessary to add salt to the diet. The recommended level, as seen in Table 51.1, is 0.5 per cent of the total ration. It can be self-fed free-choice. The work of Vestal (1946, 1947a) has demonstrated very well the value of salt in a ration composed of corn, soybean oil meal, alfalfa meal, limestone, and steamed bone meal. Vestal (1947b) also found that when half the soybean oil meal in the ration was replaced with meat and bone scrap, the pigs ate only half as much free-choice salt and that adding salt to a ration of grain and meat scrap or tankage did not improve rate or efficiency of gain.

There apparently is some concern among swine feeders about "salt poisoning" in swine. This condition is discussed in detail by Whitehair in Chapter 49. In general it is recommended that swine feeders avoid feeding materials high in salt and palatable to pigs, such as brine, whey, or slop. It is also recommended that swine always be supplied with the proper amount of salt in their rations or that loose salt be self-fed free-choice at all times. It is also good practice to provide ample trough space and fresh water in plentiful amounts for the animals.

Iron is required for the formation of hemoglobin and for the prevention of nutritional anemia in suckling pigs (see

Chapter 19) Copper is believed to be re quired along with iron for hemoglobin synthesis The iron requirement is reported to be 10–15 mg daily for the first six weeks after birth (Beeson et al , 1953) The cop per requirement has not been definitely established, but 2 mg of copper fed daily has prevented the appearance of a copper deficiency Iron and copper deficiencies are seldom seen in older swine except in areas where the soil is deficient in one or both of these elements Thus this is almost exclusively a baby pig problem Nutrition al anemia can be prevented by placing pigs on soil not deficient in these elements before the pigs are seven days old, or if this is not practical, by placing fresh, clean sod in the pen with the pigs This disease can also be prevented by drenching the pigs every other day for at least two weeks with 2–4 ml of a saturated solution of fer rous sulfate (Calderon, 1919) The often recommended practice of swabbing the sow's udder with an iron solution was not effective in Calderon's experiment.

A new method for preventing nutritional anemia in suckling pigs recently has been announced by Armour Veterinary Labora tories (1957) It is a radical departure from the oral procedures in current use This method involves the intramuscular injection of a single dose of 2 cc. of Armi dexan (a solution of a low molecular weight dextran iron compound) at one to three days of age It is claimed this treat ment will protect the pigs several weeks

Cobalt is probably essential to swine, but the nature of its function has not been clearly defined There is some question as to whether swine have a cobalt requirement when the vitamin B_{12} requirement is met. It may be that swine require cobalt only to the extent that it is present, as a con stituent, in the vitamin B_{12} that is re quired

Although manganese is known to be re quired by swine, the quantitative require ments are not known Beeson et al (1953) suggest a level of 18 0 mg of manganese

per pound of total diet as being adequate for growth and reproduction, but state that this figure does not represent a minimum requirement

Magnesium is known to be essential to swine but the quantitative requirements are not known This element is involved in bone and tooth formation and in the con trol of muscular contractions

Beeson et al (1953) report that rations containing 0 23 to 0 28 per cent of potas sium are adequate for normal growth Corn contains 0 27 per cent of the element and the other grains more, so it is not nec essary to add potassium to practical swine rations

Sulfur is required by swine in that it is a constituent of methionine and cystine and of certain vitamins Whether swine have any other requirements for this element has not been established The quantitative requirement is not known

Iodine is required by swine for the for mation of thyroxine, the hormone of the thyroid gland There are certain areas in the country where the soil is deficient in iodine In these areas it is advisable to feed additional iodine to swine, particu larly to bred sows This can be done very easily by feeding stabilized iodized salt Apparently the requirement of bred sows is about 0 1 mg per pound of total ration

The role played by zinc in swine nutri tion is not well understood but is being investigated actively at the present time Parakeratosis is a severe skin disease, or dermatitis, of swine and has been found to be aggravated by an excess of calcium in the diet (1 per cent or more) and it is generally believed that the excess of cal cium increases the zinc requirement. It has been observed that the addition of 0 01 to 0 02 per cent of zinc carbonate in many cases would prevent or cure the disease The basic mechanism of the physiological action of zinc is not under stood Parakeratosis has been reviewed by Groschke (1955) It is discussed further by Kernkamp in Chapter 50

VITAMIN REQUIREMENTS

Vitamins are very important in the proper nourishment of swine of all ages However, the highest levels are, in general required by sows and gilts during gesta tion and lactation and pigs from birth to a weight of about 70 lb The various vita mins perform a variety of functions which will be mentioned briefly

The vitamins are often classified ac cording to whether they are soluble in fat or water There are four fat soluble vita mins, namely vitamin A or its precursors the carotenes, and some related com pounds, vitamin D, vitamin E and vita min K All are required by swine but the last two are of little concern to the prac tical feeder Vitamin E is reportedly present in adequate amounts in ordinary rations built around whole cereal grains and supplemented by pasture or the feed ing of normal amounts of good quality legume hay These ingredients are good sources of the vitamin Vitamin E is re quired for normal reproduction and for the prevention, in serious deficiency, of muscular dystrophy

Vitamin K is synthesized in the intes tines of swine and ample amounts are present in green, leafy forages whether fresh or cured Vitamin K is necessary for normal clotting of blood

Vitamin A has several functions in swine It is necessary for the maintenance of the normal condition of the epithelium which lines the various body cavities the digestive tract, the respiratory tract and the urogenital tract. It is also involved in vision and is required to prevent nerve degeneration Vitamin A is required for the normal functioning of the gonads and to prevent abortions or the birth of mal formed pigs

Carotene is converted into vitamin A in the intestinal wall and to a limited ex tent in the liver This conversion is not as efficient in the pig as in some other animals, it requires 2.5 times as much carotene as true vitamin A to meet the

pig s requirement Almost all of the vita min A activity of swine rations is in the form of carotene or the compound crypto xanthine which is the yellow pigment in yellow corn Plant materials contain very little if any true vitamin A

The requirements of swine for vitamin A activity are given in Table 51 1 as milli grams of carotene per pound of total ra tion These requirements are fully met when swine are grazed on good pasture or when the usual amounts of good green legume hay are included in dry lot rations Yellow corn aids materially in meeting the vitamin A requirements

Carotene is rapidly destroyed in feeds in storage so not too much confidence should be placed in hay or corn that has been stored for a year Carotene disap pears more rapidly from ground stored feed than from feed that is not ground

When plant materials that are rich in carotene are not available vitamin A sup plements such as fortified fish liver oils or dry vitamin A supplements are available

Vitamin A is stored to a considerable extent in the liver and the fat depots of the body when the carotene or vitamin A in take is in excess of the daily requirement This stored vitamin can be used to cor rect dietary deficiencies

Vitamin D is required for the proper utilization of calcium and phosphorus Thus it is essential for maintenance growth and reproduction

Vitamin D_2 is formed by the irradiation of the plant sterol, ergosterol with the ultraviolet rays of sunlight Vitamin D_3 is formed by the effect of ultraviolet light on the animal sterol 7-dehydrocholesterol Each is equally effective in preventing vitamin D deficiency in swine When the animals are exposed to adequate sunlight as when grazing on pasture in the sum mer, they have no dietary require ent for vitamin D Under such circu ances enough of the 7-dehydrocholesterol in their skin is changed to vita in D_3 by the sunlight that their require is met.

During the winter months, when not exposed to sufficient sunlight, swine have a dietary requirement for vitamin D Usually the most practical source for winter feeding is sun cured hay Alfalfa hay cured in the field has enough vitamin D that 10 per cent of such hay in the ration will meet the requirements of all classes of swine If sun cured hay is not available, irradiated yeast is an excellent, very economical source Fortification of a swine ration with vitamin D_2 from irradiated yeast costs only a few cents per ton of feed

Green growing forage contains little if any vitamin D It is only after the plant is cut and exposed to sunlight that the forage develops vitamin D activity

The group of water soluble vitamins required by swine includes vitamin C and the so-called vitamin B complex Swine do not have a dietary requirement for vitamin C because they are able to synthesize it in their bodies

The vitamin B complex as required by swine includes thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, choline, vitamin B_{12}, biotin, folic acid, possibly vitamin B_{13} and certain unidentified factors

Thiamine is required for normal carbohydrate metabolism This vitamin is seldom a problem in swine feeding because the grains are good sources as are most of the other feedstuffs usually included in rations fed to swine

Riboflavin is a component of several enzyme systems This vitamin sometimes presents a problem in certain dry lot rations because it is present in the grains in low amounts, and the oil meals and packing house by products are not good sources Pasture crops, legume hays, milk by products distillers dried solubles, and fermentation solubles are rich in riboflavin Rations fed in dry lot should _____ _____ of one or more of th _____ to _____ adequate levels of th _____ The min is available in _____ form

Niacin, or nicot _____ serve

constituent of certain enzymes which are involved in respiration and carbohydrate metabolism

A niacin deficiency apparently can cause necrotic enteritis or bloody scours in young pigs according to Dunne et al (1949) This disease, as has been pointed out in Chapter 49, is usually associated with a filth borne organism, Salmonella choleraesuis Davis et al (1943) observed that adequate niacin intake did not prevent necrotic enteritis caused by S choleraesuis but often did aid the pig in recovering from the symptoms of the infection

The pig apparently can use extra tryptophan to synthesize niacin but cannot utilize niacin in correcting a tryptophan deficiency (Luecke et al, 1947)

Niacin is not apt to be a limiting nutrient in swine rations including a variety of common ingredients

Pantothenic acid is required by swine to prevent nerve degeneration and certain organ changes The requirements listed in Table 51 1 are 5 0 mg per pound of total ration for pigs weighing 25 and 50 lb and 4 5 mg per pound for older swine These levels are higher than those used by Barnhart et al (1954) with pigs from 25 to 100 lb live weight Levels of 2, 3, and 4 mg of pantothenic acid per pound of total ration were fed and no symptoms of pantothenic acid deficiency were observed Corn and barley are rather low in this vitamin, and dry lot rations compounded around these feeds are likely to be deficient in pantothenic acid unless liberal amounts of oats and/or alfalfa hay are included Good pasture supplies adequate levels of the vitamin

Weanling pigs weighing up to 50 lb **0.6** of pyridoxine per pound **The** for older swine vitamin is required is involved in the of amino acids distribution of pyridoxine feedstuffs, it is swine rations

Choline is usually classified with the water soluble vitamins although it is not always a dietary essential in the same sense that the others are because it can be syn thesized from other nutrients, such as methionine The requirement for 25 lb pigs, as indicated in Table 51 1, is 400 mg per pound of ration The require ments for older swine are not known Dry lot rations which contain large propor tions of corn may not contain 400 mg of choline per pound

Folic acid is required by the baby pig, but the quantitative requirements are not known Since this factor is widely distrib uted in swine feeds, a deficiency is not likely to occur

Vitamin B₁₂ is needed for the formation of hemoglobin and red blood cells It is required in the diets of young pigs in very small amounts Table 51 1 indicates that 25 lb pigs need 7 µg per pound of feed and that 50 and 100 lb pigs require 5 µg of vitamin B₁₂ per pound of feed The requirements for older animals are not known although bred sows produce heavier, stronger pigs when fed vitamin B₁₂ The vitamin seems to be of greatest importance in dry lot feeding There is some synthesis of the vitamin in the in testines of swine and it may be that this is stimulated by the consumption of fresh, green material On the other hand, swine on pasture may receive some vitamin B₁₂ from the insects and worms that they un doubtedly consume

Protein supplements of animal origin such as fish meal, condensed fish solubles, and packing house by products are good sources of vitamin B₁₂ There are also available vitamin B₁₂ feeding supplements that contain at least 1 5 mg of the vitamin per pound of supplement. Some antibiotic feeding supplements contain substantial amounts of vitamin B₁₂

Unidentified factors supplied by pasture plants, alfalfa meal, condensed fish solu bles, dried corn distillers solubles, and some other feedstuffs have been found to improve growth, feed efficiency, and brood

sow performance when one or more of these feeds have been added to rations supposedly adequate in all known nutri ents It is important, for maximum pro duction, that one or more of these sources of unidentified factors be included in ra tions for gestation, lactation, and growing pigs up to about 70 lb

FEED ADDITIVES

A number of different materials, m iny of which have no actual nutritive value, have been added to swine rations for the purpose of stimulating growth and/or in creasing efficiency of feed utilization

Antibiotics may be described as com pounds produced by microorganisms which stop or inhibit the growth of other micro organisms There are a number of anti biotics but only a few have been demon strated to produce a reasonably consistent response in growing swine Among these are Aureomycin (chlortetracycline), Ter ramycin (oxytetracycline) procaine peni cillin, and bacitracin The exact mode of action of the antibiotics in producing a re sponse in pigs is not known, although they apparently affect the microflora of the in testinal tract There is experimental evi dence to support this observation because several experiments have shown that the degree of response is related to the inci dence of subclinical disease in the herd The following effects of adding the proper level of an effective antibiotic to rations for growing and fattening pigs have been observed

1 Increase in growth rate of 10 to 12 per cent.

2 Possible increase in efficiency of feed utilization of 5 per cent

3 Greater response in younger animals.

4 Continuous feeding from the suckling period to market weight is more eco nomical than feeding to 100 lb live weight only

5 Effective reduction in incidence and severity of scouring in young pigs

6 Reduction in number of runty p gs

During the winter months, when not exposed to sufficient sunlight, swine have a dietary requirement for vitamin D Usually the most practical source for winter feeding is sun cured hay Alfalfa hay cured in the field has enough vitamin D that 10 per cent of such hay in the ration will meet the requirements of all classes of swine If sun cured hay is not available, irradiated yeast is an excellent very economical source Fortification of a swine ration with vitamin D_2 from irradiated yeast costs only a few cents per ton of feed

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The group of water soluble vitamins required by swine includes vitamin C and the so-called vitamin B complex Swine do not have a dietary requirement for vitamin C because they are able to synthesize it in their bodies

The vitamin B complex as required by swine includes thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, choline, vitamin B_{12} biotin, folic acid, possibly vitamin B_{13} and certain unidentified factors

Thiamine is required for normal carbohydrate metabolism This vitamin is seldom a problem in swine feeding because the grains and good sources as are most of the other feedstuffs usually included in rations fed to swine

Riboflavin is a component of several enzyme systems This vitamin sometimes presents a problem in certain dry lot rations because it is present in the grains in low amounts, and the oil meals and packing house by products are not good sources Pasture crops, legume hays, milk by products, distillers dried solubles, and fermentation solubles are rich in riboflavin Rations fed in dry lot should contain enough of one or more of these feeds to insure adequate levels of the vitamin The vitamin is available in crystalline form

Niacin, or nicotinic acid, serves as a constituent of certain enzymes which are involved in respiration and carbohydrate metabolism

A niacin deficiency apparently can cause *necrotic enteritis or bloody scours* in young pigs, according to Dunne et al (1949) This disease, as has been pointed out in Chapter 49, is usually associated with a filth borne organism, *Salmonella choleraesuis* Davis et al (1943) observed that adequate niacin intake did not prevent necrotic enteritis caused by *S choleraesuis* but often did aid the pig in recovering from the symptoms of the infection

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Weanling pigs weighing up to 50 lb require 0 6 mg of pyridoxine per pound of feed The requirements for older swine are not known The vitamin is required for an enzyme which is involved in the intermediary metabolism of amino acids Because of the wide distribution of pyridoxine in commonly used feedstuffs, it is not likely to be deficient in swine rations

Choline is usually classified with the water soluble vitamins although it is not always a dietary essential in the same sense that the others are because it can be synthesized from other nutrients, such as methionine   The requirement for 25 lb pigs, as indicated in Table 51 1, is 400 mg per pound of ration   The requirements for older swine are not known   Dry lot rations which contain large proportions of corn may not contain 400 mg of choline per pound

Folic acid is required by the baby pig but the quantitative requirements are not known   Since this factor is widely distributed in swine feeds, a deficiency is not likely to occur

Vitamin B_{12} is needed for the formation of hemoglobin and red blood cells   It is required in the diets of young pigs in very small amounts   Table 51 1 indicates that 25 lb pigs need 7 μg per pound of feed and that 50 and 100 lb pigs require 5 μg of vitamin B_{12} per pound of feed   The requirements for older animals are not known although bred sows produce heavier, stronger pigs when fed vitamin B_{12} The vitamin seems to be of greatest importance in dry lot feeding   There is some synthesis of the vitamin in the intestines of swine and it may be that this is stimulated by the consumption of fresh, green material   On the other hand, swine on pasture may receive some vitamin B_{12} from the insects and worms that they undoubtedly consume

Protein supplements of animal origin such as fish meal, condensed fish solubles and packing house by products are good sources of vitamin B_{12} There are also available vitamin B_{12} feeding supplements that contain at least 1.5 mg of the vitamin per pound of supplement. Some antibiotic feeding supplements contain substantial amounts of vitamin B_{12}

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sow performance when one or more of these feeds have been added to rations supposedly adequate in all known nutrients   It is important, for maximum production, that one or more of these sources of unidentified factors be included in rations for gestation, lactation and growing pigs up to about 70 lb

FEED ADDITIVES

A number of different materials many of which have no actual nutritive value, have been added to swine rations for the purpose of stimulating growth and/or increasing efficiency of feed utilization

Antibiotics may be described as compounds produced by microorganisms which stop or inhibit the growth of other microorganisms   There are a number of antibiotics but only a few have been demonstrated to produce a reasonably consistent response in growing swine   Among these are Aureomycin (chlortetracycline)   Terramycin (oxytetracycline)   procaine penicillin and bacitracin   The exact mode of action of the antibiotics in producing a response in pigs is not known although they apparently affect the microflora of the intestinal tract   There is experimental evidence to support this observation because several experiments have shown that the degree of response is related to the incidence of subclinical disease in the herd   The following effects of adding the proper level of an effective antibiotic to rations for growing and fattening pigs have been observed

1   Increase in growth rate of 10 to 12 per cent.

2   Possible increase in efficiency of feed utilization of 5 per cent.

3   Greater response in younger animals.

4   Continuous feeding from the suckling period to market weight is more economical than feeding to 100 lb live weight only

5   Effective reduction in incidence and severity of scouring in young pigs

6   Reduction in number of runty pigs

7 Results in a more uniform group of pigs at market weight

8 Increase in bloom and in some cases effectiveness in preventing an unidentified skin dermatitis on certain rations

9 Favorable response both in dry lot and on pasture

An effective level is 5 mg of antibiotic per pound of total ration or 25 mg per pound of protein supplement to be fed free choice with grain However, higher levels are often recommended Research indicates that one effective antibiotic fed at the proper level produces as much response as a combination of antibiotics The inclusion of an antibiotic in rations for bred and lactating sows has not been effective in improving production The subcutaneous implantation of antibiotic pellets in suckling pigs is not as effective in increasing weaning weights as the incorporation of the antibiotic in the creep feed for the pigs

Various arsenic compounds have been tested as additives to swine rations Two have been demonstrated to be effective in increasing rate of gain and efficiency of feed utilization of growing and fattening pigs These are arsanilic acid (4 amino phenylarsonic acid) or sodium arsanilate and 3 nitro-4 hydroxyphenylarsonic acid The degree of response depends upon the disease level' in the herd These compounds have therapeutic value in preventing and controlling swine enteritis The level of arsanilic acid to be used should be limited to not more than 90 gm per ton of total feed Arsanilic acid produces a greater response when fed in combination with an antibiotic than when fed alone Because of the possibility of an accumulation of arsenic in the edible tissues of swine, arsenic compounds should be omitted from the feed of pigs for a few days before slaughter

Surface-active agents have not proved to be satisfactory as additives to swine rations as growth promotants although only a few have been tested This work is continuing

Hoefer (1956) made reference to an experiment at Michigan State University in which a combination of streptomycin and sulfaquinoxaline added to a ration fed to growing and fattening pigs at the rate of 25 gm of each per ton of feed produced a marked increase in rate of gain

Miller *et al* (1951) observed a significant increase in rate of growth in pigs fed a fortified corn soybean oil meal ration to which 0 5 per cent of sulfathalidine had been added

It seems probable that under certain environmental circumstances in which growing and fattening pigs are hosts to intestinal microorganisms that are antagonistic to the pigs, bacteriostats included in the diet at low levels reduce the antagonistic effect and make possible a more rapid rate of gain

The effects of adding fats to rations for growing and fattening swine have been studied in several experiments (Barrick *et al*, 1953, Day *et al*, 1953, Kropf *et al*, 1954, Perry *et al*, 1953) Some of these additions have increased rate of gain and efficiency of feed utilization It seems that the value of added fat depends upon the level of fat used, the kind of fat, and the nutrient balance of the ration after the fat addition Apparently the most effective level is between 8 and 18 per cent Soybean oil and peanut oil have been somewhat less effective than beef fat, coconut oil, and commercial grease The addition of fats to a ration at a level between 8 and 18 per cent increases calorie density of the ration and changes the ratio of energy to the other nutrients For optimum results this requires a proportionate increase in levels of essential amino acids and possibly other nutrients More research is needed in this area

The early weaning of pigs (5 to 10 days) is being studied as a means of reducing the cost of production of market hogs This entails the use of milk replacers Most successful milk replacers have included a rather large proportion of dry skim milk which is quite expensive Lewis *et al* (1955) have found that baby pigs under 3 weeks of age produce amounts of proteolytic and amylolytic enzymes

which are insufficient for the adequate di gestion of dietary proteins and carbo hydrates other than those of milk These researchers have found that supplementa tion of basal diets for pigs under 3 weeks of age, in which the protein source was either soybean protein or casein, with cer tain proteolytic enzymes increased gains and feed efficiency Further research in this area may make it possible to reduce drastically or even eliminate the dry skim milk used in milk replacers for pigs

The use of hormones and hormone like materials in swine rations has received considerable attention by research workers in recent years Among these are thyro proteins, diethylstilbestrol, and methyl testosterone

The results of Braude (1948, 1950) in dicate a definite increase in rate and economy of gain when combinations of iodinated casein and stilbestrol are ad ministered orally to growing pigs As much as 680 mg of iodinated casein and 40 mg of stilbestrol were fed per pig daily It was observed that the treated animals had longer backs and legs and that they were less fat than the untreated controls

Taylor and Gordon (1955) fed growing pigs a diet containing 6 mg of stilbestrol and 0 3 mg of thyroxine per pound of feed with no increase in rate of gain or feed conversion They reported five cases of toxicity with three fatalities out of 18 treated pigs while none occurred in the controls The symptoms could be repro duced by feeding stilbestrol alone at a level of 20 mg per pound of feed

Beeson et al (1955) reported results of feeding diethylstilbestrol at a level of 2 mg per pig daily, or 20 mg per pig daily of methyltestosterone, to growing and fat tening pigs The feeding of these hormones did not improve the rate or economy of gain The stilbestrol produced increased mammary development in both barrows and gilts and caused enlarged vulvas in the gilts The feeding of methyltestosterone caused an increase in the percentage of lean cuts and reduced the percentage of fat cuts in the carcasses compared with the untreated animals The stilbestrol fed

pigs produced carcasses intermediate be tween the untreated pigs and those fed testosterone with respect to percentage of fat cuts and lean cuts Chemical analysis revealed that the testosterone fed pigs had 5 per cent less fat and 5 per cent more lean in their carcasses than the controls

Perry et al (1956) reported the results of feeding to growing and fattening pigs an average of 0–62 mg of methyltestoster one per animal daily in a free choice pro tein supplement in dry lot from a starting weight of 51 lb to a final weight of 210–220 lb A daily intake of 27 mg or more of the methyltestosterone produced a highly significant growth depression but a significantly decreased back fat thickness

Gobble et al (1957) have fed diethyl stilbestrol at three levels (75, 150, and 300 μg per pound of feed) or methyltestosterone at five levels (2 4, 6, 12, and 15 mg per pound of feed) to growing and fattening pigs, gilts being developed for the breed ing herd, and boars and barrows castrated at different ages, to determine the effect of these materials on rate and efficiency of growth carcass value and reproductive capacity in gilts intended for the breeding herd These levels of diethylstilbestrol had no effect on rate or economy of gain or carcass composition of growing and fat tening pigs slaughtered at 200 lb When this estrogenic material was fed to de veloping gilts from weaning weight to a weight of 220 lb at the 300 μg level, no effect was observed on age at puberty or length of estrous cycle or period. However, reproductive capacity was reduced some what Developing gilts fed 1 mg of methyl testosterone per pound of feed from wean ing weight to 220 lb exhibited delayed puberty, irregular estrous periods and decreased reproductive per cycles, and those which farrowed had dif formance Those which farrowed had dif ficulty at parturition because of subnormal size of the birth canal Barrows and gilts fed levels of 2 or 4 mg of methyltestoster one per pound of feed from weaning to 200 lb showed little effect of the treat ment on rate or economy of gain and carcass composition Boars and unilateral cryptorchids fed 15 mg of testos erone per

pound of feed from weaning and slaugh tered at about 220 lb showed subnormal testicle size but had seminal vesicles, pros tate glands, and bulbo urethral glands which were about normal in size Loin roasts and chops from these boars, fed for more than 100 days, did not have the characteristic boar odor during and after cooking but were somewhat more coarse in texture of lean than untreated barrows of similar age Data also were obtained relative to the effect of the 6 and 12 mg levels on rate and economy of gain, cer tain carcass characteristics, and reproduc tive organs and glands of barrows and gilts No consistent increase in carcass value was observed in gilts Depending on length of feeding period, both levels in creased carcass value in barrows without significantly decreasing rate of gain

It is known that 15 mg of methyltestos terone per pound of feed fed to barrows from weaning to 215 lb will significantly increase the percentage of lean cuts and decrease the percentage of fat in the carcass but at the same time significantly decrease *the rate of gain*

The cost of methyltestosterone at present is prohibitive for use in practical rations and optimum levels and methods of feed ing have not yet been determined

FEEDING THE BROOD SOW AND GILT

The importance of the feeding of brood sows and gilts should be emphasized for at least two reasons the cost of the feed given to these animals constitutes a sub stantial portion of the total cost of pro ducing market hogs, and the size and weight of the litter at farrowing and the subsequent performance of the pigs pro duced are materially affected by the feed ing of the sow herd Thus, in order to realize a maximum return from the enter prise, it is necessary to feed rations which are balanced nutritionally and are eco nomical The value of pasture or liberal amounts of high quality legume hay in these rations should be emphasized The amount of feed fed is also very important

Underfeeding of pregnant animals and lactating animals is harmful to the off spring Overfeeding is wasteful and may reduce the number of pigs farrowed and weaned Excessive fatness is to be avoided in both sows and gilts because this de creases their ability to farrow large litters of strong pigs Overfat sows are less likely *to raise the pigs they farrow because they* are clumsy and often lazy

Sows and gilts should be fed liberal amounts of balanced rations for 2 or 3 weeks prior to the breeding season, if pos sible It is thought that this increases the number of eggs ovulated After breeding, the amount of feed given should be re duced to about 1 25 lb of feed per 100 lb live weight for sows and about 1 75 lb per 100 lb live weight for gilts The latter need a higher level of feeding because they are still growing The nutrient require ments of pregnant gilts can be broken down into four fractions   requirements for maintenance, growth, products of con ception, and preparation for lactation The last three fractions involve the storage of nutrients The pregnant sow does not have a requirement for growth Pregnancy itself does not impose any particular hard ship on the sow or gilt because the amounts of nutrients stored in the products of con ception are not large

During gestation, sows and gilts are usually hand fed although they may be self fed if the ration is bulky enough to prevent their becoming too fat Rations to be self fed to pregnant sows and gilts should contain 12 to 15 per cent or more of crude fiber, and the condition of the animals should be carefully watched so that the proper gains are made Exercise is considered by many to be a factor in the production of strong, vigorous pigs at birth

Usually the pregnant female is placed in her farrowing pen or stall a few days before she has her pigs This ordinarily means a restriction of exercise with a pos sibility of constipation The substitution of wheat bran for one third to one half of

the regular ration prevents this The consistency of the droppings should be watched carefully

The sow needs no feed for 12 hours before and after farrowing If she is rest less and seems hungry, a handful of bran on her water will help to quiet her

After farrowing, the amount of feed given the sow should be limited If the sow is fed too much too soon after the pigs are born, she will produce more milk than the pigs can take, with caked udders and scouring pigs the result Feed the sow about 2 lb of feed the second day after the pigs are born and gradually increase the amount until she is on full feed by the time the pigs are 10 to 12 days old

It is advisable to self feed a sow nursing 6 or more pigs Such a sow, if producing well, often will not be able to eat enough feed to produce the milk and maintain her body weight Self feeding increases feed consumption The ration fed the sow during lactation should be balanced and palatable and should be fairly low in fiber The fiber content should be limited to a maximum of 8 per cent Sows particularly during lactation, should have free access to clean, fresh water A few days before weaning, the amount of feed given the lactating sow should be reduced by one half to two-thirds This will tend to reduce the milk flow so that when the sow is separated from her pigs the danger of caked or damaged udder sections will be reduced The sow's feed should be limited after weaning until her udder is dried up

Baby pigs should have access to a suitable ration in a creep, in addition to the sow's milk, as soon as they will eat dry feed The creep ration should be well fortified with vitamins and minerals have protein of high quality, be concentrated and palatable The use of excellent pasture for suckling pigs is highly desirable High quality protein supplements of animal origin are quite valuable in a pig starter or creep ration The palatability of the mixture can be improved by in

cluding 10 per cent of cane or beet sugar and by pelleting the ration The feeding of an effective antibiotic to suckling pigs is indicated Because of the complex nature of many recommended pig starters it is often economical for the smaller producer to purchase a commercial starter from a reputable feed manufacturer

The herd boar should be fed so as to keep him in a vigorous thrifty condition, neither too thin nor too fat Pasture should be provided when possible because of the nutrients it provides and because of the increased opportunity for exercise Rations suitable for pregnant sows and gilts are suitable for mature boars and young boars, respectively (see Table 51 1)

The amount of feed given the boar is usually increased just prior to and during the breeding season Boars sometimes go off feed When this happens, particularly during the breeding season the use of rather expensive feedstuffs to improve feed consumption is justified Milk, rolled oats, raw eggs, and sugar or molasses are especially well liked

The feeding of growing and fattening pigs from weaning to market weight is a very important factor in the financial success of swine production It is in this area that the swine feeder has a great opportunity to reduce his cost of production The nutrient requirements for market swine of various weights are given in Table 51 1 The formulation of rations for these animals should be considered Following are the steps to be followed in one method of ration formulation

1 Identify the class of swine to be fed
2 Select the appropriate set of nutrient requirements
3 Select suitable ingredients so as to make the ration
   a balanced nutritionally
   b palatable and safe
   c economical
4 Determine the amount of each ingredient to be used except for the low protein cereal grain and one high protein supplement (During this step the vita

min and fiber levels are adjusted, protein quality is provided for, and an antibiotic is added)

5 Adjust the amount of cereal grain and high protein supplement so as to have a mixture which supplies the correct level of protein

To demonstrate the use of this procedure the formulation of a ration for a 50 lb pig to be fed for slaughter in dry lot may be used as an example The requirements for 50 lb market stock given in Table 51 1 are used In steps three and four a knowledge of the nutrient content of the various available feedstuffs is necessary The list of ingredients selected could be yellow, dent corn, meat and bone scrap, 50 per cent protein grade, soybean oil meal, solvent process, high quality

alfalfa hay, ground, antibiotic feed supplement, vitamin B$_{12}$ feed supplement, and salt All these ingredients are safe palatable, and economical

Meat and bone scrap included at a level of 5 per cent improves the protein quality of the mixture and helps to raise the protein level, adds materially to the calcium and phosphorus contents, and adds water soluble vitamins, especially vitamin B$_{12}$ Ten per cent of high quality alfalfa hay meets the requirement for carotene and vitamin D, adds calcium and protein, and helps to raise the water soluble vitamin levels except for vitamin B$_{12}$ The vitamin B$_{12}$ supplement is used to increase the amount of this vitamin supplied by the meat and bone scrap to the point that the requirement is met If the vitamin B$_{12}$

TABLE 51 3

EXAMPLES OF SWINE RATIONS USING THE COMMON GRAINS

	Corn	Barley	Wheat	Oats	Wheat Mids	Ground Alfalfa Hay	36% Hog Suppl *	Total
During Gestation (hand fed)	1,000			500		100	400	2,000
During Gestation (self fed)	800			400		500	300	2,000
During Lactation (hand or self fed)	1,100			400		100	400	2,000
During Lactation (hand or self fed)	1,100			200	200	100	400	2,000
Growing & Fattening (self fed)	1,500						500	2,000
Growing & Fattening (self fed)	1,300			200			500	2,000
Growing & Fattening (self fed)	800	700					500	2,000
Growing & Fattening (self fed)	600		600	400			400	2,000
Growing & Fattening (self fed)	1,000			200	400		400	2,000

* For example of a 36% all purpose hog supplement, see tables below Most commercial hog supplements containing about 36% protein can be used in these rations

A 36% HOG SUPPLEMENT

Ingredient	Amount
Meat scrap tankage or fish meal (60%)	400 lb
Alfalfa meal (16%)	400 lb
Soybean oil meal	800 lb
Cottonseed or linseed oil meal	300 lb
Minerals (see table at right)	100 lb
Irradiated yeast	0 5 lb
Niacin †	12 gm per ton
Riboflavin	4 gm per ton
Pantothenic acid	8 gm per ton
Vitamin B$_{12}$	40 mg per ton
Antibiotic (Aureomycin or Terramycin)	50 gm per ton

MINERAL MIXTURE

Ingredient	Amount
Iodized salt	25 lb
Dicalcium phosphate or steam bone meal	25 lb
Ground raw limestone	50 lb
Ferrous sulphate	2 0 lb
Copper sulphate	0 1 lb
Cobalt sulphate or carbonate	0 1 lb
Manganese sulphate	0 2 lb
Zinc carbonate	0 8 lb

Note This mineral mixture can be made available free choice to all classes of swine

† Vitamins and antibiotics in amounts indicated are necessary to obtain optimum performance These vitamins and antibiotics are included in most commercial hog supplements If the farmer wishes to mix his own supplement, these vitamins and antibiotics must be obtained from a feed dealer

potency of the supplement is 1 5 mg per pound then 0 25 per cent will be needed The result of adding the antibiotic feed supplement is obvious If the supplement supplies 5 gm of antibiotic per pound then 0 1 per cent will be needed to supply a level of 5 mg per pound Corn is the major source of energy, and the soybean oil meal is used to supply protein quantity and quality, some phosphorus and certain water soluble vitamins To avoid any possibility of a salt deficiency 0 5 per cent of salt is added The amounts of corn and soybean oil meal must be adjusted to provide a level of protein of 16 per cent

Set up the ration in the following manner formulating on the basis of 100 lb

Ingredient	Pounds	Protein Supplied (lb)
Corn		
Soybean oil meal		
Meat and bone scrap	5 00	2 49
Alfalfa hay	10 00	1 60
Antibiotic feed supplement	0 50	
Vitamin B₁₂ supplement	0 25	
Salt	0 50	
	15 85	4 09

In order to have 100 lb of mixture it is necessary to add 84 15 lb of corn and soybean oil meal This amount of these two ingredients will have to supply 11 91 lb of protein The correct proportions of corn and soybean oil meal can be calculated in the following manner

Let $x =$ lb of corn needed

Let $y =$ lb of soybean oil meal needed

Then $x + y = 84\ 15$

One pound of corn supplies 0 087 lb of protein One pound of soybean oil meal supplies 0 457 lb of protein

Then $0\ 087x + 0\ 457y = 11\ 91$

When the two equations are solved simultaneously it is found that 71 76 lb of corn and 12 39 lb of soybean oil meal will complete the desired 100 lb of mixture and will provide a protein level of 15 99 per cent This ration provides also the following amounts of the various required nutrients that need be considered calcium 0 71 per cent phosphorus 0 56 per cent carotene 3 0 mg per pound vitamin D 91 International Units per pound riboflavin 1 4 mg per pound niacin 11 mg per pound vitamin B₁₂ 5 25 μg per pound and pantothenic acid 3 53 mg per pound The ration is balanced except for pantothenic acid which is deficient by 1 47 mg per pound This deficiency can be corrected by adding calcium pantothenate at the rate of 147 mg per 100 lb of feed in an appropriate premix using ground corn as a carrier

The principles involved in formulating the above ration may be used in compounding rations for any class of swine Rations for swine on good pasture need not include hay or a B vitamin supplement with the possible exception of vitamin B₁₂ and can be somewhat lower in protein than the levels indicated in Table 51 1

Table 51 3 gives examples of some swine rations If it is desired to self feed the grain and protein supplement free-choice it is possible to use a protein supplement similar to the one given in Table 51 3 either custom mixed or purchased from a reputable feed manufacturer

For more complete details on swine feeding refer to *Feeds and Feeding* by F B Morrison published by the Morrison Publishing Company Ithaca New York and to *Swine Production* by W E Carroll and J L Krider published by the Mc Graw Hill Book Company New York

REFERENCES

ASSOCIATION OF AMERICAN FEED CONTROL OFFICIALS. 1948 Official Publication.

ARMOUR'S ANALYSIS. 1957 Vol 5

BARNHART C E CATRON D V., QUINN L Y., AND ASHTON G C 1951 Partial nutritional requirement of weanling pigs fed a purified ration Jour Anim Sci 13 9 4

BARRICK E R BLUMER T N BROWN W L SMITH F H. LOVE S B LUCAS H L AND STEWART W A 1953 The effects of feeding several kinds of fat on feed lot performance and carcass characteristics of swine Jour Anim Sci 12 899

BEESON W M 1956 Mineral and amino acid requirements of swine Feedstuffs. 23 12.

Beeson, W. M., Andrews, F. N., Perry, T. W., and Stob, M.: 1955 The effect of orally administered stilbestrol and testosterone on growth and carcass composition of swine. Jour. Anim. Sci 14 475.

———, Crampton, E. W., Cunha, T. J., Ellis, N. R., Luecke, R. W: 1953. Nutrient requirements for domestic animals No. 11. Nutrient requirements for swine. Nat. Res. Council Publ 295

Bohstedt, G 1955 Calcium and phosphorus in swine rations Proc. Cornell Nutr. Conf for Feed Manufacturers.

Braude, R. 1948 Stimulation of growth in pigs by iodinated casein and stilbestrol Nature, London 161 856.

——— 1950 Stimulation of growth in pigs by iodinated casein and stilbestrol. Brit. Jour. Nutr. 4 138

Calderon, C. A 1949 Suckling pig anemia in relation to nutritional anemia. M. S Thesis, Pa. State Coll, Unpublished.

Carroll, W. E., and Krider, J. L.: 1953. Swine Production, 2nd ed McGraw Hill Book Co, New York

Chapman, H. L., Jr., Kastelic, J., Ashton, G. C., and Catron, D V.: 1955. A comparison of phosphorus from different sources for growing and finishing swine. Jour. Anim Sci. 14 1073

Davis, G. K., Hale, E. B., and Freeman, V. A: 1945 Response of pigs given large doses of Salmonella choleraesuis to sulfaguanidine, nicotinic acid, thiamine and pyridoxine Jour. Anim Sci 2 138

Day, B N., Anderson, G C., Johnson, V. K., and Lewis, W. L.: 1953 The effect of a high fat ration on swine gains and carcass quality. Jour. Anim. Sci. 12 944.

Dunne, H. W., Luecke, R W., McMillen, W. N., Gray, M. L., and Thorp, F., Jr.: 1949 The pathology of niacin deficiency in swine. Amer. Jour. Vet. Res 10 351.

Gobble, J. L., Miller, R. C., Sherritt, G W., and Dunne, H W: 1956 Soft phosphate with colloidal clay as a source of phosphorus for growing and fattening pigs. Pa. Agr. Exp. Sta. Bull 609

———, ———, Ziegler, P T., and Dunne, H. W: 1957. Pa. Agr. Exp. Sta. Unpublished data

Grossiaup, A. C 1955 Parakeratosis, a type of dermatitis in pigs Proc. Nutr Council, Amer. Feed Manufacturers Assn (May), p 10.

Hanson, L. E 1951 Forty years of progress in swine feeding Feed Age 1 20.

Hoffer, J. A: 1956. Advances in swine nutrition. Proc. Cornell Nutr. Conf., p. 44.

Jordan, C. E., Kennington, M. H., Plumlee, M. P., and Beeson, W. M.: 1956. Phosphorus supplements for swine. Ind. Agr. Exp. Sta. Mimeo A. H. 182.

Kropf, D H., Pearson, A. M., and Wallace, H. D.: 1954. Observations on the use of waste beef fat in swine rations Jour Anim Sci 13 630

Lewis, C. J., Catron, D V., Liu, C. H., Sierk, V. C., and Ashton, G. C.: 1955. Enzyme supplementation of baby pig diets. Jour. Agr. Food Chem. 3 1017.

Luecke, R. W., McMillen, W N., and Thorp, F., Jr.: 1947. Further studies on the relationship of niacin and protein in swine nutrition. Jour. Anim Sci. 6 483.

Mertz, E. T., Beeson, W. M., and Jackson, H. D.: 1952. Classification of essential amino acids for the weanling pig Arch Biochem and Biophysics. 38 121.

Miller, R C., Gobble, J. L., and Krins, L. J.: 1954. Response of pigs to feeding of vitamin B₁₂, streptomycin and sulfathaludine. Proc. Soc. Exper. Biol. and Med. 78 164.

Morrison, F. B., and Fargo, J. M.: 1922. Efficient rations for pigs. Wis. Agr. Exp. Sta. Bull. 352, p 21

Perry, T W., Beeson, W. M., and Mohler, M. T: 1955. Adding animal fat to swine rations. Ind Agr. Exp. Sta. Mimeo. A. H. 116.

———, ———, Andrews, F. N., and Stob, M: 1956. The effects of various levels of orally administered methyl testosterone on growth and carcass composition of swine. Jour. Anim Sci 15 1003

Plumlee, M P., Kennington, M. H., and Beeson, W. M: 1955 Utilization of phosphorus from various sources by growing fattening swine. Jour. Anim. Sci. 14.1220.

Taylor, J H., and Gordon, W. S.: 1955 The effect of feeding a diet containing stilbestrol and thyroxine to growing pigs with special reference to the toxicity of stilbestrol. Vet. Rec. 67 48

Vestal, C. M. 1946 Pigs need salt — never let them get hungry for it. Ind. Agr. Exp Sta. Mimeo. A. H. 20

——— 1947a. Prevent salt hunger in hogs. Ind. Agr. Exp. Sta. Mimeo A. H. 23.

——— 1947b The kind of supplement fed influences the amount of extra salt required in a hog ration. Ind. Agr. Exp. Sta. Mimeo A. H. 23

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CHAPTER 52

Swine Management

Successful hog producers skillfully direct all phases of the enterprise Those who make the most money practice good management by carefully controlling feeding breeding sanitation shelter, and general care of their hogs at all stages of growth They try to do each task at the proper time and in such a way as to get the best performance per unit of labor, feed, and investment in stock and equipment In actual practice many compromises are necessary, but more money is probably lost through poor management than through any other major aspect of hog production

The importance of good feeding and nutrition is stressed in Chapters 49 and 51 Good feeding must be coupled with a good breeding system, sound sanitation and disease control, proper shelter, and careful management of each phase of the swine raising cycle

BREEDING

The art of breeding is old, but the science of breeding is relatively new The results of more than 20 years of scientific swine breeding research are available (Craft, 1953) In general, these results tend to parallel the history of the development of hybrid corn Future research will doubtless answer many questions that can not be answered satisfactorily at this time

The performance of swine varies as a result of differences in *heredity* and *environment* and the joint effects of the two Heredity is based on genes that are passed from parent to offspring Thus improvements in heredity are passed on to the offspring When the environment of the pig is improved by the provision of better feeds, more comfortable quarters and protection against disease, an immediate improvement in the animal is evident, but none of this improvement is passed on to the offspring by genes.

To understand recommended breeding systems, definitions of the common terms used in swine breeding work are helpful

Common Terms Used in Swine Breeding Work

Selection is the choosing of the males and females to be kept as breeding animals to produce the next generation Selection may be based on a single characteristic, such as type, prolificacy, growth rate or feed efficiency, but usually it involves a combination of these and other characteristics Selection is more effective for some traits than for others.

Heritability is the fraction or per cent of variation in a trait that is caused by hereditary differences in individuals It represents the amount of the select ed advantage in breeding stock that will on the average, be transmitted to the next

generation Thus selection is effective in changing a trait that has a high heritability for example a trait that is affected to a fairly large extent by heredity and to a fairly small extent by environment

Outbreeding is the mating of animals within a breed that are unrelated The genetic background of an outbred herd is changed quite frequently by introducing into the herd boars that are unrelated to the sow herd

Crossbreeding is a more extreme form of outbreeding in which animals of different breeds are mated In a rotation cross breeding system three or four breeds of boars are used in sequence and the cross bred gilts are bred to a boar of the next breed in rotation

Inbreeding is the mating of animals that are more closely related than the average of their breed An example of the closest possible sort of inbreeding would be the breeding of a boar to his litter mate sister or to his dam An inbred line is developed by mating several generations in a group closed to outside blood Usually two gener ations of brother sister matings or four or five generations of half brother–half sister matings are needed to develop an inbred line Linebreeding is a very mild form of inbreeding in which the blood of a cer tain ancestor is concentrated by using his descendants in the breeding herd

Inbred lines have been developed by close matings within a herd of (1) a pure breed or (2) a crossbred foundation from two or more pure breeds An illustration of pure breed matings is the inbred lines developed within the Poland China Duroc, Landrace Yorkshire and other breeds at the various experiment stations

The procedure of developing inbred lines from a crossbred foundation has been used extensively at the Minnesota Station and by the U S Department of Agricul ture Some of these inbred lines have been expanded and registered as inbred breeds by the Inbred Livestock Registry Associa tion Following are some of the inbred lines that were developed from a cross bred foundation

Inbred Line	Breeds Used in Foundation (approximate percentage of total)
Beltsville No 1	Poland China (25%) Land race (75%)
Beltsville No 2	Duroc (32%) Hampshire (5%) Landrace (5%) Yorkshire (58%)
Maryland No 1	Berkshire (38%) Landrace (62%)
Minnesota No 1	Landrace (55%) Tamworth (45%)
Minnesota No 2	Poland China (60%) York shire (40%)
Minnesota No 3	Beltsville No 2 (6%) Gloucester Old Spot (31%) Large White (11%) Poland China (20%) Minnesota No 1 (5%) Minnesota No 2 (4%) San Pierre (9%) Welsh (14%)
Montana No 1	Hampshire (45%) Landrace (55%)
Palouse	Chester White (65%) Land race (35%)
San Pierre	Berkshire (50%) Chester White (50%)

Hybrid, as the term is generally accepted by animal breeding specialists means a combination of inbred lines The hybrid is produced by crossing two or more inbred lines usually from different breeds The terms linecross and incross are often used to indicate a cross of inbred lines Line crossing can be done either within or be tween breeds but the term is often re stricted to the crossing of inbred lines with in a breed, whereas the terms hybrid and incross are commonly used when the in bred lines that are crossed belong to dif ferent breeds For example a seedstock producer of hybrid boars might cross two inbred lines, such as Beltsville No 1 and Maryland No 1, to furnish hybrid boars for the first cross in a farmer s herd He might cross Minnesota No 3 with an in bred Duroc line to furnish the second hy brid boar in the farmer s rotation crossing program And then he might cross two inbred lines, such as Minnesota No 2 and Beltsville No 2, to produce hybrid boars for the third cross in the rotation

Hybrid vigor is the term applied to the increase in performance that results when unrelated breeds or lines are crossed Crossbreds express hybrid vigor because they receive unlike genetic material from the dam and the sire

Summary of Swine Breeding Research

Selection is an effective means of changing type in hogs For example, the meat type characteristics of a 'lardy' herd can be improved rapidly by using boars that have a minimum of backfat thickness and by selecting the gilts in the herd that have the least backfat thickness Carcass traits such as length of body, backfat thickness, percentage of fat cuts, and loin lean area, have high heritabilities falling in the range of 40 to 60 per cent

Present methods of selection do not seem to be effective in producing further improvement in litter size and brood sow productivity in superior purebred strains Also, the heritability of number of pigs farrowed or weaned per litter and of litter weaning weight appears to be low — about 10 to 15 per cent

Present methods of selection are mildly effective in improving rate or efficiency of gains Only about 15 to 25 per cent of the difference in these traits between pigs is due to inheritance The rest is due to such things as feeding, management, and disease

Inbreeding usually reduces performance As inbreeding goes up, it seems to be most detrimental to brood sow productivity, pig survival, and sexual maturity, but growth rate is also reduced to some extent In breeding, however, seems to have little effect on carcass characteristics or on feed efficiency

The traits that are affected most unfavorably by inbreeding and that respond least to selection (for example, brood sow productivity and pig survival) produce the greatest amount of hybrid vigor when breeds are crossed Crossbreeding tends to produce some hybrid vigor for growth rate and to cause earlier sexual maturity in both males and females, but improvements in meat type are made primarily by selection Crossbreeding seems to give the greatest boost when the breeds that are crossed come from widely different genetic backgrounds Outbreeding within a breed also produces hybrid vigor but not to the extent as the crossing of breeds

Briefly, research results indicate that some of the most important production characteristics are difficult to improve further by selection as it is now practiced These same characteristics deteriorate greatly by inbreeding, but are helped by crossbreeding and in certain instances are helped further by skillfully combining in bred lines

Recommendations for Commercial Hog Producers

Commercial hog producers should follow a crossing program because the results in nearly all experiments have shown cross breds to be superior to non-crossbreds

One of the simplest methods of crossing to produce market hogs is to rotate three or four breeds or lines of boars Each boar can be used until the gilts from his litters go into the breeding herd Then these gilts are bred to the next breed or line in the rotation The older boar may be kept for breeding the dams of these gilts as long as they are retained in the herd

Commercial hog producers may use any one of the following kinds of rotations (1) purebred boars (2) inbred boars, (3) hybrid boars, or (4) a combination of two or more in one rotation

The following examples of rotations of purebred boars are given only as suggestions other combinations may be equally good or better

Example 1 — Landrace, Poland China Duroc

Example 2 — Yorkshire, Hampshire, Duroc

Example 3 — Berkshire, Landrace, Spotted Poland China To follow the rotation of purebred boars given in Example 1 above, the hog producer would mate a Landrace boar to his present sows When the daughters of the Landrace boar were selected as herd replacement gilts, he would buy a Poland placement gilts, he would buy a Poland

China boar to breed to them When the daughters of the Poland China boar were introduced into the breeding herd, he would secure a Duroc boar to breed to them Daughters of the Duroc boar would be bred to a Landrace boar and the rotation continued

Here are some suggested rotations of in bred boars

Example 1 – Maryland No 1, Minnesota No 2 Minnesota No 3

Example 2 – Beltsville No 1, Beltsville No 2 San Pierre

Example 3 – Minnesota No 1, Minnesota No 2, Minnesota No 3

In a rotation of hybrid or incross boars, the procedure would be the same except that each boar would be a cross between two or more inbred lines Some companies selling hybrid boars make known the in bred lines they use to produce each hybrid boar other companies do not reveal this information In some instances the hybrid boar producer crosses inbred lines that have a common breed in their background

Here are some examples of rotations using a combination of purebred and in bred boars

Example 1 – Maryland No 1, Duroc, Minnesota No 2

Example 2 – Landrace, San Pierre, York shire

Example 3 – Beltsville No 1, Hamp shire Yorkshire

The effectiveness of a rotation cross-breeding program will depend upon (1) whether the boars come from herds where there has been effective selection for meat type, growth rate, feed efficiency, and brood sow productivity, and (2) whether the strains used in the rotation have high combining (nicking) ability Much of the current swine breeding research is concerned with identifying outstanding lines that have combining ability or with developing lines that combine well in a crossing program

Two plans are being followed to improve traits where nicking is important. One is to make many inbred lines and test them in crosses to see which ones nick particularly well The other is to establish two strains that have already proved to cross well and then progeny test the members of each strain by crossing them to the other strain Then only the parents with the best crossbred progeny should be used to perpetuate the pure strain This process is called reciprocal selection The results of current and future research will probably tell which plan is best for the swine industry

Testing Programs

Both seedstock producers and commercial hog producers conduct programs to identify outstanding stocks To make better breeding stock available to his customers the seedstock producer must effectively test and select for improvement in *meat type, growth rate, feed efficiency,* and *brood sow productivity* in his herd

Commercial hog men are demanding records on boars available for purchase These are some of the characteristics they want in the boars they buy from seedstock producers

1 A backfat thickness of not more than 1 3 inches at 200 lb when full fed
2 Weigh 200 lb at five months of age
3 Produce a pound of gain on no more than 3 3 lb of feed
4 Come from a herd that has had at least eight pigs raised per sow

MEASUREMENTS TO USE IN TESTING PROGRAMS

The producer will usually be interested in testing for one or more of the following economically important characteristics meat type, growth rate, feed efficiency, and brood sow productivity Simple measures are needed that are reasonably easy to make and that give reliable estimates of the trait. Then, if these results are used in an effective selection program, progress will be made Of course, improvement will be most rapid for the traits that have the highest heritability

Meat type can be measured in a number of ways The animals can be appraised visually for such characteristics as length backfat thickness and other points of body conformation believed to be correlated

with a high percentage of lean cuts Slaughter information, such as carcass backfat thickness, percentage of lean cuts area of loin eye muscle, weight of the closely trimmed ham, and specific gravity of the carcass, is an excellent measure of the carcass qualities of the animal slaughtered This information can be applied to close relatives Use of the live probe technique (Hazel and Kline, 1952) permits an indirect estimate of the carcass qualities of boars and gilts to be used as breeding stock since the correlation between the live backfat probe and the percentage of lean cuts is quite high To live probe for backfat thickness, a small incision is made though the skin with a sharp knife about two inches off the midline of the back at the shoulder, back and loin Then a small steel ruler is pressed though the fat at each of these points The ruler stops when it hits the pork chop muscle The average of three readings indicates the backfat thickness of the animal With another device the lean meter,' a needle is pushed through the backfat to determine backfat thickness A meter reading shows when the pork chop muscle is reached Most breeders probe when their pigs weigh about 200 lb, but a probe adjustment table can be used to adjust to an equivalent probe at 200 lb

Growth rate is commonly measured by obtaining a five or six month weight on each pig Since it is not usually convenient to weigh each pig as it reaches five or six months of age, weight adjustment tables can be used to obtain an equivalent five or six month weight In either live probing or weighing at five or six months of age, it is important to be able to identify each pig by a system of ear notching and to keep a record of the birth date of each litter tested

Selecting by growth rate from weaning to market weight may be more effective than selecting by weight at five or six months of age, because heritability estimates are higher for the former than for the latter

Feed efficiency can be measured directly by keeping feed and gain records on individual pigs litters or groups of pigs from the same sire This approach is used at central testing stations and by a few progressive seedstock producers Feed efficiency also can be measured indirectly by taking advantage of the high correlation between growth rate and feed efficiency Because of the close relation between the two selection for growth rate is, on the average automatic selection for feed efficiency

Brood sow productivity can be measured best by litter weaning weights The National Association of Swine Records has adopted basic requirements for an all breed production registry Witnessed farrowing and 56 day weighing reports must be sent to the breed record association on nominated litters Production registry requirements are as follows

1 A mature sow must farrow and raise eight or more pigs to a 56 day weight of at least 320 lb

2 A first litter gilt must raise the same number of pigs to a 56 day weight of at least 275 lb

3 Sows qualify for production registry after producing two production registry litters

4 To qualify as production registry sires boars must sire five qualified daughters or fifteen daughters that have produced one production registry litter

Some of the breed record associations also have sow production testing programs based on 21 day or 35 day litter weights rather than 56 day weights The various state swine improvement associations have litter testing programs based on litter weaning weights

Testing programs can be classified as (1) on the-farm testing and (2) central station testing Each approach has its advantages and disadvantages In the on the farm program, large numbers can be tested and the data collected under the breeders own farm conditions The information that is obtained should be used to improve selection in the breeders own herd It should not be used to compare the herd with other herds where conditions may be quite different. The coordination of a community educational program for swine improve

ment with individual testing programs may cause promotional and publicity benefits to accrue to the seedstock producers.

The program for certified meat hogs adopted by the National Association of Swine Records is an example of on-the-farm testing. It is a three-point program based on (1) production registry, (2) rate of gain, and (3) carcass quality.

A *certified litter* is qualified as follows: (1) The litter must qualify for production registry. (2) Two pigs from the production registry litter must weight 200 lb. or the equivalent at 180 days. Weights must be off truck weights of the pigs when delivered at the cooperating slaughter station. The pigs are to be delivered for slaughter at a weight between 180 and 230 lb. Equivalent 180-day weight is calculated by adding 2 lb. for each day under 180 days old and deducting 2 lb. for each day over 180. (3) The same two pigs from the litter must meet the carcass standards given in Table 52.1.

Each pig is tattooed when weighed off-truck. The loin area is calculated by means of planimeter tracings of the loin eye made on parchment paper. The loin is broken at the tenth rib. The carcass length is measured from the front of the first rib where it joins the vertebra to the front of the aitch bone. The backfat is an average of three measurements taken opposite the first rib, the last rib, and the last lumbar vertebra. Actual backfat thickness is measured to the outside of the skin and at a right angle to the back.

A *certified boar* is one that has sired five litters that qualify as certified litters. These five litters must be out of five different sows, not more than two of which are full sisters or dam and daughter.

A *certified mating* is the repeat mating of a boar and a sow that have produced a certified litter.

Another example of on-the-farm testing is the Illinois Swine Testing Extension Project. Designed for use by both commercial and purebred breeders, it is essentially a "probe and weigh" program to obtain adjusted 180-day weights and live backfat probes on a 200-lb. basis in addition to any litter weaning weight data that are being collected.

Central station testing has been carried on for many years in Denmark. In this country this approach to swine improvement has been used recently by local or county swine herd improvement associations and by a few colleges and breed record associations.

At the central testing station the aim is to provide as nearly as possible the same environmental conditions by using identical shelter and equipment and by having each pig or group of pigs eat the same ration, occupy the same amount of space, and go through the test at the same time. This plan makes possible more valid comparison of the herds represented at the station than can be obtained by comparing on-the-farm records of the same herds. It is particularly helpful to the boar purchaser. It gives the seedstock producer publicity, and if he submits several pigs or groups of pigs, it also gives him a basis for comparing them.

Test stations are rather expensive to operate, and the number of pigs that can be tested is often somewhat limited. This method of testing will probably be most useful in providing demonstrations for educational programs involving hog producers and a means of comparing herds that rank high in on-the-farm testing.

SANITATION

There is no substitute for sanitation in efficient hog production. More money is

TABLE 52.1
CARCASS STANDARDS FOR CERTIFIED MEAT HOGS

Weight	Minimum Loin Area	Minimum and Maximum Length	Minimum and Maximum Backfat Thickness
(lb.)	(sq. in.)	(in.)	(in.)
180–200	3.50	28.5–32.0	1.0–1.6
201–215	3.75	29.0–32.5	1.1–1.65
216–230	4.00	29.5–32.0	1.2–1.7

lost through failure to follow a sanitation system than through any other form of mismanagement All four of the following steps in sanitation must be taken if the hog program is to be effective

The farrowing houses must be cleaned A steam cleaner or boiling hot lye water (1 pound of lye to 30 gallons of water) should be used A power sprayer can be effective if used persistently until all dirt and foreign material are removed When the house is dry it should be sprayed with a good disinfectant The cleaned farrowing house and equipment are to be left idle for at least three weeks before each farrowing season These sanitation breaks will help to prevent the build up of disease

The sow must be washed with warm water and soap before putting her into the clean pen or stall to farrow It is a good idea to spray her at this time to help control lice and mange

The sow and pigs should be given a clean ride to clean pasture or a clean concrete drylot Both sows and pigs must be kept off contaminated lots

Clean pasture should be provided by raising at least one cultivated crop on the area between pig crops *Concrete lots should be kept clean* by steam cleaning (or cleaning thoroughly with water under pressure) and disinfecting the area between pig crops

Worming sows before the breeding season or in early pregnancy may help to produce healthy pigs by breaking up the roundworm cycle

MANAGEMENT DURING THE BREEDING SEASON

Proper management during the breeding season is essential in obtaining a high conception rate in the sow herd and large litters of healthy pigs

The Boar

Hog producers often make the mistake of economizing on the number of boars they purchase to sire their pig crops Over working boars may reduce the quality and sperm concentration of semen The immediate result will be a lower conception rate smaller litters and a longer farrowing period The final result will be management problems and lower profits

The number of sows that a boar can settle during a given breeding season will depend on the age and individual libido or sexual drive of the boar the method of mating and other factors involving both boars and sows including management and control of disease

In hand mating the boar is brought to each sow that is in heat for mating or vice versa whereas in pen mating the boar is allowed to run with the sows during part or all of each day Each method has certain advantages and disadvantages In hand mating more sows can be bred with the same number of boars a record can be kept of each mating and the expected farrowing date can be calculated for each sow Calculating farrowing dates may help to prevent losses at farrowing time

Boars vary considerably in their capacity to breed during a given breeding season The recommendations given in Table 52 2 can be used as a guide by breeders A good herdsman with active boars will be able to exceed the values given in this table

The boar pig should be well grown and at least eight months old before being used for breeding Inbred boars may show less sex drive and reach sexual maturity at a later age than outbred or hybrid boars

The Female

Various factors such as age weight amount of inbreeding breed and number

TABLE 52.2
RECOMMENDED NUMBER OF SOWS PER BOAR FOR A GIVEN BREEDING SEASON

Length of Season	Hand mating		Pen mating	
	Boar 1 yr	Mature Boar	Boar 1 yr	Mature Boar
2 weeks	15	25	10	15
4 weeks	25	35	15	5
6 weeks	35	45	3	30
8 weeks	45	60	5	35

ment with individual testing programs may cause promotional and publicity benefits to accrue to the seedstock producers

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216–230	4 00	29 5–32 0	1 2–1 7

lost through failure to follow a sanitation system than through any other form of mismanagement All four of the following steps in sanitation must be taken if the hog program is to be effective

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Worming sows before the breeding season or in early pregnancy may help to produce healthy pigs by breaking up the roundworm cycle

MANAGEMENT DURING THE BREEDING SEASON

Proper management during the breeding season is essential in obtaining a high conception rate in the sow herd and large litters of healthy pigs

The Boar

Hog producers often make the mistake of economizing on the number of boars they purchase to sire their pig crops Over working boars may reduce the quality and sperm concentration of semen The immediate result will be a lower conception rate smaller litters and a longer farrowing period The final result will be management problems and lower profits

The number of sows that a boar can settle during a given breeding season will depend on the age and individual libido or sexual drive of the boar the method of mating and other factors involving both boars and sows including management and control of disease

In hand mating the boar is brought to each sow that is in heat for mating or vice versa whereas in pen mating the boar is allowed to run with the sows during part or all of each day Each method has certain advantages and disadvantages In hand mating more sows can be bred with the same number of boars a record can be kept of each mating and the expected farrowing date can be calculated for each sow Calculating farrowing dates may help to prevent losses at farrowing time

Boars vary considerably in their capacity to breed during a given breeding season The recommendations given in Table 52 2 can be used as a guide by breeders A good herdsman with active boars will be able to exceed the values given in this table

The boar pig should be well grown and at least eight months old before being used for breeding Inbred boars may show less sex drive and reach sexual maturity at a later age than outbred or hybrid boars

The Female

Various factors such as age weight amount of inbreeding breed and number

TABLE 52.2

RECOMMENDED NUMBER OF SOWS PER BOAR FOR A GIVEN BREEDING SEASON

Length of Season	Hand mating		Pen mating	
	Boar Pig	Mature Boar	Boar Pig	Mature Boar
2 weeks	15	45	10	15
4 weeks	25	35	15	5
6 weeks	35	45	25	45
8 weeks	45	65	25	35

of heat periods (in the gilt), affect the number of eggs ovulated during the estrous cycle of female swine After ovulation has occurred, litter size is further affected by such things as physical abnormalities of the reproductive tract, disease, nutrition, and probably other factors that are not well understood at present

The gilt should be well grown and at least eight months of age before being bred for the first time Ovulation rate tends to increase with each heat period for the first three heat periods after the gilt reaches puberty This increase provides the basis for the general recommendation that gilts should not be bred during the first two heat periods

Sows usually come into heat three to four days after weaning pigs at the usual weaning age of six to eight weeks The hog producer therefore can exercise a considerable amount of control over the length of the next farrowing season by deciding when to wean each litter

Multiple mating of each sow during a heat period is often recommended as a means of increasing conception rate and litter size Craig et al (1955) studied the effect of single mating on the first or second day of heat and of double mating on the first and second day to the same boar on rate of settling of 402 females bred in seven seasons They reported a conception rate 1½ percentage points higher for double mating than for single mating (78 vs 64 per cent respectively) A second service 12 to 24 hours after the service on the first day of heat has been most beneficial in breeding seasons when boar fertility is low

These same workers also studied the effect of time of single mating and the effect of double mating on litter size in 197 litters farrowed in four seasons They reported a nonsignificant overall difference of one pig per litter in favor of breeding on the first day over the second day of heat and differences of 0 6 pig and 0 1 pig in favor of double mating over mating on the second and first day of heat, respectively More research is needed, but these results cast some doubt upon the sound

ness of usual recommendations that sows be bred on the second rather than the first day of heat when 'boar power' is limited

Breeding Season Tips

These things done before the breeding season starts will help to increase the possibility of getting good results

1 Blood testing both boars and females for brucellosis and leptospirosis

2 Removal of the tusks from the boar

3 Increasing the daily feed of the boar so that a gain in weight will result

4 Mating the boar to a few extra sows or gilts The first service after a period of inactivity is often an infertile one Each young boar pig should be hand mated to a sow before turning him out with the sow herd for pen or pasture mating

5 Increasing the feed for sows a week or so before the breeding season starts and continued feeding at the higher rate to the end of the period Self et al (1955) showed the value of "flushing" gilts during the breeding season These workers reported that the greatest number of normal embryos in gilts slaughtered at the 25th day of gestation resulted from a sequence of hand feeding at two thirds the full fed rate from about 70 days of age to puberty, full feeding until bred during the second heat period, and then hand feeding at two thirds the full fed rate during the first 25 days of gestation

6 Providing the boar with adequate shelter and if possible a dirt or pasture exercise lot Ordinarily it is better not to pen boars next to the sow lot The excitement caused by being near sows in heat may cause boars to 'rant and go out of condition On the other hand, it might help to pen a sluggish boar near the sow herd

7 Boars of the same age or of different ages can be penned together if they are observed and supervised carefully until they establish a social order Boars placed together during hot weather must be watched carefully

8 Varying feeding to fit conditions Usually the ration fed to bred gilts will be satisfactory for the boar Between breeding

seasons it is advisable to keep the mature boar on good legume pasture and feed at the rate of one to one and one fourth pounds of feed daily per hundred pounds of live weight

These tips apply when hand mating is practiced

1 The boar may be trained to use a breeding crate during his first breeding season

2 He should be fed after the service rather than before Most herdsmen prefer to hand feed in a trough and to wet the feed with water or a milk product Wheat bran and rolled oats are often included in the boar ration

3 Patience and avoidance of any action that will cause the boar to be wary are important

4 Sows and gilts should be bred during the first day of heat If enough boars are available, each sow should be bred again 24 hours later

5 A record of each mating should be kept so that a farrowing date can be scheduled for each female

6 The boar should be observed carefully for signs of disease or loss of appetite, and appropriate action taken if either occurs

MANAGEMENT DURING GESTATION
Desired Gain

An average daily gain of about three fourths pound by bred sows or gilts will provide adequately for the growth of the gilt and development of the fetuses and also provide a body reserve for a lactation period of one to six weeks If sows or gilts are thin when bred and must nurse litters beyond six weeks or be on a limited feeding program during lactation, a gain of at least one pound a day during gestation is recommended

Terrill et al (1955) compared the gestation performance of 65 bred sows and gilts fed three levels of a well fortified corn soybean oil meal ration Three, 4 5, and 6 0 lb of ration were fed daily to bred gilts and bred sows on scanty rye pasture during the winter The sows barely maintained their body weight when fed 3 lb

of feed daily whereas the gilts gained one third pound a day on the same feed in take Feeding 4 5 lb of feed per head daily to gilts or sows produced the desired daily gain of about ¾ lb during the last 90 days of gestation, and the farrowing performance was satisfactory

Self-Feeding Versus Hand Feeding

Bred gilts and sows may be either self fed or hand fed Self feeding requires less labor, but a bulky, fibrous ration must be fed in order to keep the energy intake low enough to prevent the bred females from getting too fat More feed is usually wasted with the self feeding method, and the proportion of bulky feeds in the ration must be changed if the sows are not gaining as desired

Hand feeding usually takes less feed, and the entire group of bred females can easily be observed at feeding time Feeding the entire daily allowance at one time each day seems to be just as satisfactory as twice a day feeding

Cutting Feed Costs for Bred Sows and Gilts

Attempts to reduce the feed cost per live pig farrowed have centered around (1) making maximum use of pasture to furnish energy and other nutrients, (2) feeding silages, and (3) self feeding low cost rations containing appreciable quantities of inexpensive fibrous feeds such as ground corn cobs or hay

Research at Illinois, Indiana, Iowa, and Minnesota has shown that, with proper supplementation, good corn or grass silage can make up a large part of the ration for sows and gilts during gestation Gilts and sows may eat from 8 to 15 lb of silage per head daily

An Illinois test (Terrill et al, 1953) showed that a ration consisting of 700 lb of ground corn cobs, 600 lb of ground yellow corn, 200 lb of alfalfa meal, and 500 lb of protein supplement was adequate for feeding bred sows

Excellent ladino clover pasture can help to reduce gestation feed costs. In tests conducted at Illinois (Terrill et al, 1954),

sows lost weight at the rate of ¼ lb a day when restricted to excellent ladino clover pasture and minerals during the last 70 days of gestation Nevertheless, they farrowed large litters of apparently normal pigs averaging 2 7 lb at birth Feeding 2½ lb of corn per head daily in addition to minerals to bred gilts on ladino clover pasture produced gains of ½ lb per head daily

SAVING BABY PIGS

Raising more pigs to market age is the quickest way to increase profits in the hog business The following management tips should help to get more pigs to market

The sow should be fed gestation rations that contain enough protein, minerals vitamins, and other nutrients which experiments have shown are needed to produce sound pigs These rations are to be started before mating and fed during the gestation period

A bulky, somewhat laxative ration may be fed the week before and the week after farrowing, but a concentrated non bulky ration can be self fed successfully during this period when sows are turned out of farrowing stalls for a period of only 1 to 1½ hours twice daily for feeding

Farrowing stalls are used to save space, reduce the activity of the sow, and prevent her from crushing the pigs Farrowing stalls are recommended over farrowing pens with guard rails Appropriate equipment for feeding and watering can be provided in the front of each stall, or the sow can be turned out twice a day to a pen or feeding platform to eat from a self feeder and drink from an automatic waterer The latter procedure reduces the amount of labor required to clean manure out of the farrowing stall area and also gives the sow exercise Farrowing stalls reduce the need to attend sows at farrowing Many hog producers who formerly acted as "midwives" at farrowing time now place sows in farrowing stalls to farrow unattended except for routine checks or assistance when it is needed.

The following ration or one similar to

it can be used to self feed sows the week before and after farrowing

Feed	Pounds
Ground yellow corn	600
Ground oats	600
Wheat bran	600
Sow supplement (35% protein)	200
Total	2 000

If a farrowing pen is used, it should have a clear floor area 6 by 6 or 6 by 7 feet. Large sows will need more space It is wise to equip the pen with guard rails that project about 8 inches above the floor and 8 inches from the wall at the sides and back If possible, heat should be supplied for baby pigs A heat pad, heat lamp, or heated floor area protected from the sow will be satisfactory If heat is not supplied, a pig hover will conserve the body heat of the pigs, help to protect against drafts, and give some protection from the sow

As soon as pigs are born, the navel stub should be daubed with tincture of iodine Needle teeth need not be clipped unless the pigs fight excessively If the teeth are clipped, care must be exercised to avoid injury to the gums Chilled or weak pigs may be fed one or two teaspoons of corn sirup diluted with two parts of water every two or three hours These pigs need supplementary heat. If the sow is slow in coming to milk, the weak pigs may be given artificial milk Extra pigs or orphan litters can be raised on artificial milk if good management and sanitary methods are used Litters can be evened by transferring pigs from large litters to small ones Pigs should be ear notched to help identify gilts from the most productive litters

Nutritional anemia in pigs kept on concrete or wooden floors can be prevented by an iron dextran injection at birth or by sprinkling a solution of 1 lb of ferrous sulfate in 3 quarts of water on a chunk of uncontaminated sod and keeping it in the pen where the pigs can root at it. This treatment should be started before the pigs are one week old

Another effective way of preventing nutritional anemia is to give each pig an iron

pill or a "squirt" of iron sulfate solution once or twice a week for three weeks or a little longer

A *pig starter ration* should be fed to suckling pigs from the time they will eat until the pigs weigh 20 pounds, and then a good complete mixed ration should be fed until they weigh at least 40 lb

A number of systems are used in raising pigs to 40 lb weight Hog producers who leave the pigs on the sow until they are 5 to 8 weeks of age usually feed a pig starter ration Some use a highly palatable *suck ling pig prestarter* (usually containing 20 to 22 per cent protein and appreciable amounts of sugar or molasses) to get the pigs eating well and then switch to a regu lar pig starter (usually 18 to 20 per cent protein) Others use a regular pig starter throughout the suckling period

Producers who wean pigs as early as two to three weeks of age (6–10 lb) usually feed a well fortified *early weaning pre starter* (about 24 per cent protein) for at least one week before switching to a starter ration containing about 18 per cent pro tein

Boar pigs should be castrated at one week of age They are easily handled and suffer very little setback at this time

Pigs should be vaccinated for cholera when they are four to six weeks of age If a killed vaccine is used, the pigs should be 10 weeks old Attenuated live virus vaccines can be used before weaning

Sows and pigs can be sprayed with lin dane or benzene hexachloride solution to control lice and mange A solution of 0 25 per cent of the gamma isomer of benzene hexachloride is very effective

The pigs should be weaned by the time they are eight weeks old Those that are thrifty, eat well, and weigh 20 lb can be weaned at five weeks With careful management and an excellent early wean ing prestarter ration, pigs weighing 6 10 lb can be weaned as early as 3 weeks of age

MANAGEMENT OF EARLY-WEANED PIGS

Some of the possible advantages of early weaning, along with certain limitations the hog producer should consider are as fol lows

1 *Early weaning can cut labor needs* by reducing the handling of feed bedding and manure below that needed for sows nursing litters

2 *It can save space* because more pigs can be cared for in the same space when sows are removed

3 *It will save sow's feed* because heavy milking sows eat lots of feed — 12 to 18 pounds a day If, however, the prestarter ration is quite expensive, this extra cost may offset part or all of this advantage

4 *It may permit sows to be rebred or sold sooner after farrowing*, although most sows cannot be successfully rebred for 30 or more days after farrowing

5 *It may help to reduce loss in weight of the sow*

The big disadvantage of an early wean ing program is that the average hog pro ducer does not realize the skill and careful management that are essential for its suc cess The younger and smaller the pig at weaning, the greater the attention that must be given to details of sanitation, en vironment, and disease control Proper equipment is also very important in han dling very young pigs

The following management tips should prove helpful in the early weaning of pigs

1 In most cases pigs should not be weaned before they weigh 10 pounds Weight and condition are better criteria than is age

2 The floor space to be allowed per pig up to four weeks of age is four square feet and for the next three or four weeks eight square feet.

3 A temperature of 75–80° F should be provided for one- and two-week-old pigs Solid wall pens will help to prevent drafts

4 Pigs should be grouped according to size and weight, and no more than 20 pigs of the same size put into the same pen

5 A well fortified ration in pig sized self feeders that permit easy access to the feed, and clean, fresh water must be pro vided at all times

6 All the steps in a good sanitation program should be followed The com

bined use of farrowing stalls with early weaning will keep death losses low, save space, and save labor

WEANING-TO-MARKET MANAGEMENT

For economical production, the crowding of hogs or the wasting of space must be avoided The shade, shelter, and equipment needs for a particular herd can be determined by consulting Table 52 3

If the pigs are confined from weaning to market and self fed, 10 square feet of feeding floor space (in addition to sleeping space) should be allowed each pig Each pig will need 15 square feet of feeding floor space if fed from troughs

One automatic watering cup should be provided for every 20 pigs (an automatic waterer with two openings is considered as 2 cups) The waterer should hold at least 25 gallons in summer and 15 gallons in winter for every 10 pigs

During hot weather, sanitary hog wallows may be provided Up to 50 pigs can be accommodated in 100 square feet of wallow Hog wallows can be placed in the

TABLE 52 3
Space Needs of Growing Finishing Swine

Item	Weaning to 75 lb	76–125 lb	126 lb to Market Size
Sleeping space or shelter per pig, sq ft.			
summer	7	9	12
winter	6	8	10
Pigs per linear foot of self feeder space (or per hole)			
on dry lot	4	3	3
on pasture	4–5	3–4	3–4
Per cent of feeder space for protein supplement			
on dry lot	25	20	15
on pasture	20–25	15–20	10 15
For hand feeding or hand watering running feet of trough per pig (fed from one or both sides)	¾	1	1½

sun, but they must be near shade or shelter and near the self feeders

Pigs varying widely in weight should not be permitted to run together Ordinarily the range in weight should be no more than 20 per cent above or below the average

Wormy pigs should be treated as indicated in Chapter 29

Pasture Versus Dry Lot

Interest in raising hogs on concrete dry lot is increasing, and it will probably continue to increase, particularly among producers who wish to specialize in hogs and who use modern buildings and labor saving equipment efficiently the year round The concrete dry lot feeding program is particularly adapted to an owner operator who has above average management skill and modern buildings and equipment that can be used the year round to the maximum degree consistent with good disease control and sanitation

On diversified farms with legumes as an integral part of the crop rotation, hog producers will continue programs that take advantage of the sanitation and labor saving benefits of a pasture program

The feed saving benefit of pasture lies mainly in protein supplement, not in grain An acre of pasture may save about 1,500 pounds of supplement for growing pigs However, an acre of highly productive land may yield a greater return in growing high profit crops than in use as a hog pasture

Pasture feeding is particularly recommended for the tenant or the small to medium sized operator who wants to have only a small investment tied up in buildings and equipment.

Complete Rations Versus Corn and Supplement Free Choice

Results of most experiments show that pork can be produced at less cost with free-choice feeding than with complete ration feeding However, especially on pasture, small pigs on a complete ration make faster gains For best gains of pigs on pasture, it

is advisable to feed a complete ration up to 70 pounds in weight before shifting to a free choice feeding program

MARKETING

The price of hogs is determined by the *supply* of hogs and pork products and the *demand* of consumers for pork products Local marketing conditions as well as the over all economic trend in price levels modify the market price of hogs

Monthly trends in hog prices and hog marketings from 1947–53 are shown in Table 52 4 Receipts of market barrows and gilts are low in July and August, in crease through December, drop off in February, increase somewhat in March as fall farrowed pigs are sold, and then de crease steadily to another low the next July and August

Prices for market barrows and gilts show an inverse relationship to marketings Highest average prices occur in July and August when marketings are lowest Prices decrease through December and remain fairly steady from January to April Fluctu ations from this general trend occur, par ticularly in certain years, but, in general, the monthly trends in hog prices can be

summarized as a cycle with two peaks and two valleys, because patterns in supply correspond to farrowing schedules fol lowed by hog producers in the hog raising regions, i e the production of spring pigs and fall pigs followed by marketing six to eight months after farrowing

Prices of sows are influenced by the sup ply of market barrows and gilts and their price Sow marketings are highest in the months of June, July, and August Market prices for sows are highest in August and September, when receipts of market bar rows and gilts are low and when barrow and gilt prices are high In general, to keep down feed costs sows should be sold as soon after they wean their last litter as is feasible But an attempt should be made to sell sows in August or September if other factors permit some choice in sow market ing dates

FARROWING SCHEDULES
One Litter a Year

Hog producers on the one litter a year system usually plan their hog operations to fit in best with the equipment labor, pas ture, and feed supplies available during the year This system is used most in the northern and western area of the corn belt The hog producer sells the sows after they have weaned their pigs To complete the cycle, he saves back herd gilts from the crop of market hogs and breeds them to farrow at about one year of age In areas where the winters are quite cold, mud is a problem in the spring, and if equipment is limited, farrowing is often delayed until late spring or early summer Hog pro ducers using this system like it because they can use cheaper equipment than for early spring farrowing and can usually schedule farrowing for the period when weather is less severe Also, they can make greater use of pasture in the summer and the new corn crop in the fall and can produce pigs of about the right size to go behind feeder cattle in the fall months A few producers follow a one litter a year system but schedule farrowing to take place during the late summer or early fall

TABLE 52 4

INDEX OF MONTH TO MONTH MARKETINGS OF HOGS
AND PRICES RECEIVED BY FARMERS IN THE
UNITED STATES (1947–53)*

Month	Barrows and Gilts		Sows	
	Mar ketings	Prices	Mar ketings	Prices
January	137	94	72	91
February	104	96	52	98
March	108	95	52	96
April	97	93	52	95
May	92	98	74	100
June	80	103	148	101
July	64	113	200	106
August	58	113	180	110
September	74	110	110	113
October	106	102	84	107
November	131	92	86	90
December	149	91	90	87

*U S D A Agricultural Marketing Service Re ports

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Two Litters a Year

Raising two litters a year rather than one permits greater use of equipment, labor, and capital throughout the year As hog production increased in the corn belt, hog producers bred gilts or sows to farrow in the early spring or fall and then, after they had weaned these litters, rebred them for a second pig crop six months later They used the same equipment for each pig crop and attempted to get as many pigs as possible on the market before the next crop arrived

When keen observers noted that hog prices usually broke when the big runs of spring or fall pigs hit the market, more hog producers began to farrow earlier in spring and fall Many hog producers who had farrowed pigs in March and September gradually changed to farrowing in February and August, and some attempted to beat the price break by farrowing in January and July The result was a shift in the price cycle to the left, i e, the peaks and valleys of prices occurred earlier in the year than formerly But earlier farrowing in most instances required better buildings and equipment and a source of heat for baby pigs, and thus production costs were increased to some extent

Three Litters a Year

Some producers farrow three times a year, using a combination of the one and two litter a year systems One herd is handled on the two litter a year basis, and the same equipment is used for both spring and fall litters Another herd is handled on the one litter a year basis The farrowing is scheduled for late spring or early summer (May or June) with simple, low cost equipment, minimum labor needs, and maximum use of pasture The early summer pigs are often limited fed to some extent and marketed when prices strengthen after the first of the year With this system a careful manager can increase hog returns over the two-litter a year program

Four or More Litters a Year

As the hog industry moves toward greater specialization, an increasing number of producers use multiple farrowing to make more efficient use of equipment and to spread marketings throughout the year This plan places less emphasis on trying to hit the high market price and more emphasis on cutting costs of production With increased multiple farrowing, the present cyclic marketing and price pattern can be evened out to some extent, but it requires excellent management, sanitation, and disease control, plus a fairly even supply of labor the year round

Most multiple farrowing programs are multiples of two litter a year herds, although some are multiples of one litter a year herds The latter plan provides an income tax advantage because a higher percentage of sales each year would come from breeding animals than when other than a one litter a year system is used Thus the maximum income could be reported as capital gain and the minimum as ordinary income

An example of a four litter a year program would be in maintaining two herds, each on a two litter a year basis For example, in one herd the females would be bred to farrow in February and rebred to farrow in August of each year, whereas in the second herd the females would be bred to farrow in May and rebred to farrow in November The four farrowing seasons would be equally spaced throughout the year A producer could use one modern farrowing unit for each farrowing and still have time for a cleanup and 'sanitation break' between farrowings He could use one set of growing finishing facilities twice a year for each herd if he provided an ample margin of safety in the form of extra space to handle the slow growing pigs

An example of a six litter a year program would be three herds, each on a two litter a year basis For example, in one herd the

females would be bred to farrow in February and rebred to farrow in August In the second herd farrowing would occur in April and October, and in the third herd farrowings would be planned for June and December Here again the farrowings would be equally spaced throughout the year to permit maximum use of farrowing facilities Only the very best managers should, however, attempt to carry out a six litter a year program Others should first gain experience on a less specialized program

In planning a hog management schedule and equipment needs, the following five units should be considered

1 Bred sow unit — where pregnant females can stay from breeding season to farrowing time

2 Farrowing unit — where farrowing takes place

3 Pig nursery unit — where the pigs are raised until they reach six to eight weeks of age

4 Growing unit — where the pigs are raised from six to eight weeks of age until they reach about 100 lb in weight

5 Finishing unit — where the pigs are finished from 100 lb to market weight

The bred sow unit may be either movable equipment located on pasture as much of the year as possible or centrally located dry lot facilities Management practices during the gestation phase may involve a low cost feeding program making maximum use of pasture or silage and hand feeding or self feeding an inexpensive bulky ration

The farrowing unit on a specialized hog farm might be a modern farrowing barn complete with farrowing stalls, radiant heating for baby pigs, and other special features On a less specialized farm it might be a pull together house equipped for farrowing, a series of individual houses concentrated at a central point or a horse,

dairy, or poultry structure converted to use at farrowing time

The pig nursery unit on a highly specialized hog farm might be an early weaning barn or wing of a farrowing barn On a less specialized farm it might be an open shed facing south on a concrete strip with pens for groups of sows and litters equipped with pig brooders and automatic feeders and waterers Or it might be movable houses for sows and litters located on pasture Often the farrowing unit and the nursery unit are combined i e a farrowing barn or movable houses on pasture may serve for the entire period from farrowing to weaning The growing and finishing units are commonly combined and the same equipment is used from weaning to market time

MANAGEMENT PROGRAMS TO MAXIMIZE HOG RETURNS

Hog returns can be maximized by (1) increasing selling price, (2) lowering production costs, or (3) changing to a specialized program that is uniquely fitted to the operator and his farm

Increasing the Selling Price

Farrowing should be scheduled and a feeding program planned so as to have hogs ready to hit the market peaks, unless in so doing the increase in production costs offsets the advantage gained by timely marketing

The selling price of hogs can be increased by producing a higher quality hog and receiving recognition (price advantage) for the improved product Greatest progress can probably be made by making improvements in breeding, i e, selecting meat type breeding stock As breeding improvements are made and maintained and as greater price differentials are paid on the basis of quality, interest in feeding hogs for improved carcass quality will increase With this end in view, much of the emphasis in current research is center

ing on the effects of energy intake, protein intake, and management practices on carcass characteristics

Selecting a market is important in increasing the selling price of hogs Hog markets and market reports should be studied On the basis of all available information, the market that seems most likely to yield the highest net return for the hogs should be selected

Selling at the proper market weight also offers an opportunity to increase the average selling price of hogs Each drove should be ' topped out at frequent intervals as the hogs approach a market weight of 200–220 lb Gains beyond these weights are more expensive and carcass grade decreases as the fat content of the carcass increases

Lowering Production Costs

Production costs can be lowered by making improvements in breeding, feeding, management, and disease control as outlined in this and other chapters of this book

Labor costs usually account for 7 to 8 per cent of the costs of raising hogs It takes about 20 man hours of labor to raise 1 litter, or 1 to 1½ man hours per 100 lb

Labor costs can be cut by putting the following ideas into use (Hardin, 1952)

1 *Concentrated farrowings* Farrowing time is a crucial and time-consuming period There is little advantage in spreading it over several weeks Extra sows or gilts and plenty of boar power can be used to concentrate the farrowing season within a short period

2 *Good farrowing facilities* It is easier to care for sows and litters at farrowing time in multiple or central houses than in individual houses

3 *Automatic or semi-automatic water supply* Pipe lines or large tank wagons can be used, and the trip to the field fitted in with other chores

4 *Automatic feed handling* Feed should be handled in bulk, and a system set up that requires a minimum number of moves

When possible, gravity along with large capacity feeders should be used

5 *Small but important jobs done on time* It is easier to castrate a one week old pig than one weighing 50 lb Other small but important jobs including vaccinating, cleaning houses and equipment, spraying for external parasites, building fences, moving pigs (or sows) at weaning time, and providing shade and shelter should not be delayed

Specialized Programs Uniquely Fitted to the Man and His Farm

If it is desirable to shift to a specialized hog program uniquely fitted to personal talents or to the local area, one of the following plans might be considered

OPERATING A PIG HATCHERY

Pig hatcheries are considered a desirable part of the hog industry because there is often a good demand for thrifty weaned pigs But the hatchery business has been held back by disease difficulties and supply, demand, and price problems Hatcheries will probably be most successful on the fringe of the corn belt, where small grains and pasture can supply most of the feed and where labor, land, and building costs can be kept low These advantages give the skilled hog man in this area an opportunity to specialize

PRODUCING BREEDING STOCK FOR SALE

The potential market for tested growthy, meat type boars is practically unlimited In the United States, commercial hog men use about 500,000 boars each year in producing about 100 million market hogs The size of this enterprise furnishes ample opportunity for breeders who wish to produce hybrid, inbred, or purebred seedstock for sale Other breeders may wish to become associate producers for purebred, hybrid, or inbred seedstock firms

Other specialized hog programs might include enterprises that are planned to utilize waste products (garbage, bakery

goods, poultry offal, milk by products, etc)

As the swine industry moves toward greater specialization, an increase may be seen in contract production and feeding of hogs, with integrated effort on the part of seedstock suppliers, feed companies pack ing companies and hog producers

REFERENCES

CRAFT, W A. 1953 Results of swine breeding research U.S D A Circ No 916

CRAIG, J V , NORTON, H W , RIO, P R AND LASLEY J F 1955 The effect of day and fre quency of mating on conception rate and litter size in swine Jour Anim Sci 14 1178

HARDIN, L S 1952 Labor saving in the hog lot. Hog Annual Farm Quart p 227

HAZEL, L N AND KLINE, E A 1952 Mechanical measurement of fatness and carcass value of live hogs Jour Anim Sci 11 313

SELF, H L , GRUMMER, R H , AND CASIDA, L E 1955 Effects of various sequences of full and limited feeding on the reproductive phenomena in Chester White and Poland China gilts Jour Anim Sci 14 573

TERRILL, S W , BECKER, D E , EDWARDS, R M NESHEIM M C AND JENSEN A H 1951 Ladino clover studies with bred gilts and sows Ill Agr Exp Sta AS 382

————— —————, AND NESHEIM, M C 1953 Grass legume silage for bred sows Ill Agr Exp Sta AS 326

————— JENSEN, A H , AND BECKER D E 1955 Cutting costs in feeding bred gilts and sows Ill Agr Exp Sta AS 417

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CHAPTER 53

Control and Elimination of Swine Diseases Through Repopulation With Disease-Free Stock

There are two fundamental reasons for control of disease in domestic animals These are (1) to reduce reservoirs of diseases transmissible to man and (2) to produce livestock more economically When human health is concerned, cost of eliminating a disease becomes secondary When human health is not a factor, sheer economics dictates the costs of control of a disease among livestock populations

Control of hog cholera and swine erysipelas has been undertaken because the severe losses which may be incurred among swine are costly (see Chapters 7 and 22) Evidence of these diseases is tangible Only recently has much attention been paid to the relatively intangible effects of a number of diseases chronic in character and associated with a retarded rate of growth and in efficient use of feed Definite evidence of inefficiencies due to chronic diseases, such as virus pneumonia of pigs, swine dysentery, and atrophic rhinitis, has been reported in the past few years by Betts and Beveridge (1953), by Betts et al (1955b), by Young et al (1955b), and by Shuman and Earl (1956) These authors emphasize the adverse economic effects of disease on the cost of pork production and the importance of eliminating diseases as herd problems

One of our most effective demonstrable means of control of animal diseases has been isolation and destruction of infected animals and their known contacts on the

farms The following basic points outline this approach (1) quarantine of premises where the outbreak occurs (2) disposal of infected and exposed animals by slaughter and burial or burning, (3) cleaning and disinfection of premises and all equipment and (4) testing the infectivity of the premises by restocking with susceptible animals It has been through use of these principles that several outbreaks of foot and mouth disease have been contained and eradicated (Mohler, 1938) The same principles and approach were used to bring vesicular exanthema under control following the epizootic which was so dramatically touched off in the summer of 1952 (Mulhern, 1953)

ELIMINATION OF SINGLE DISEASES

Less drastic means of disease control based on the same general principles, have been used to reduce greatly the incidence of brucellosis among our swine populations (Hoerlein et al, 1954) Contact of diseased animals with animals free of brucellosis is brought to a practical minimum by tests for the removal of reactors from within a herd. Since brucellosis progresses at a relatively slow rate through contacts, removal of reactors is helpful in eliminating the disease Brucellosis may be eliminated from good lines from the adult separating the young stock from the adult stock and maintaining the young stock away from other swine Separation of the

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young from their dams may be delayed until the general weaning age of 6 to 8 weeks Spread of brucellosis from the dam to the pig during the suckling period is hindered by immunity derived by the pig from suckling his dam Pigs are separated from their dams before this immunity wanes to a level where infection spreads from adults to the young If colostral immunity has not been adequate to prevent brucellosis in the pigs, subsequent blood tests on the pigs may show reactors These reactors are culled immediately and tests are made 30 and 60 days later on the remaining stock Only stock which does not react on two or more successive tests is considered free from brucellosis It should be retested annually

Stock free from virus pneumonia of pigs (VPP) is obtained in a manner similar to that used for brucellosis except that there is no blood test which can be used to detect infected animals Elimination of VPP is dependent upon the dam's not being a disease spreader during the contact period with her pigs Spread is limited from litter to litter by use of isolated A houses in pasture during the farrowing and suckling of the pigs Litters of pigs in which there has been no coughing or other signs of VPP by 8 weeks are weaned and kept isolated from other stocks Representative barrows from each litter may be sacrificed and examined for presence of possible VPP lesions (see Chapter 5 for descriptions of typical VPP symptoms and lesions) Litters passing these tests as free of VPP may be combined to constitute a new breeding herd (Barber et al, 1955, Betts et al, 1955a, Betts et al, 1955b)

The successful elimination of atrophic rhinitis (abbreviated as AR) by use of similar principles has also been reported (Shuman and Earl, 1956) Pigs were caught at birth on sterile canvas towels and were immediately removed to a new and clean environment There they were raised on artificial diets away from other swine By minimizing contact of the pigs with the sow or her environment, pigs were prevented from contracting AR although their dams were from an infected herd with evidence of many carrier gilts and sows The immediate removal of the new born pig from its dam's environment is essential for elimination of AR Colostral immunity to AR is not adequate to prevent spread of AR from the dam to the pigs in her litter during the suckling period Thus it is more difficult to eliminate AR from a herd than brucellosis AR may not be as easily detected at 6 to 8 weeks as VPP The sacrifice of barrows for careful examination of the nasal turbinates would not assure absence of AR

The differences in the methods described above are in the manner in which clean stocks are obtained The manner and its complexity are dependent upon the characteristics of the disease The single objective, once the stock is free of the disease for which elimination was designed, is to keep the stock clean The disadvantages of these methods are (1) their tediousness and (2) the fact that in each instance the objective is to eliminate a single disease

MULTIPLE ELIMINATION OF DISEASE

Effective means of obtaining pigs at birth which are free from disease have been developed which overcome the dilemma presented by methods which eliminate one disease at a time Disease free pigs are obtained from their dam 2 to 4 days prematurely by hysterectomy (Young et al, 1955b), by cesarotomy (Whitehair and Thompson, 1956), or by hysterotomy (Hoerlein et al, 1956) Pigs may also be caught at natural birth in sterile canvas bags (Young and Underdahl, 1951, 1953), in sterile basins (Done, 1955), or on sterile canvas towels (Shuman et al, 1956) The procedures by which pigs are freed without transversing the birth canal are preferable Chance of the pigs becoming infected while passing through the birth canal or from feces or flatus is eliminated

Hysterectomy means removal of the womb In the technique to be described, the gravid uterus is used as an encasement

for transport of the disease free unborn pigs into a clean environment so they may be born without exposure to swine diseases Figure 53 1 diagrammatically presents features of this method as a disease eradication and control principle

The dam from which pigs are to be removed by hysterectomy is hoisted by both hind legs She is lowered head first into a 55 gallon open top steel drum filled with carbon dioxide gas dispersed from crushed dry ice The dam is allowed to inhale carbon dioxide for one minute and an abdominal incision is made immediately It is made through the abdominal wall on the midline into the peritoneal cavity just cephalad to a point opposite the posterior most teats The incision is extended 12 to 15 inches cephalad while forcing the gravid uterus and other viscera aside The opera

PROCURING PIGS
1 STERILE HOOD
2 BARREL FOR CO2 GAS
3 WATER LOCK—PASS
  UTERUS INTO HOOD
4 HEATER & AIR FILTER
5. WORKING AREA
6. DOOR FOR REMOVAL
  OF PIGS

TRANSPORTING
1.WOODEN BOX
2. CANVAS TOP
3. ZIPPER & FLAP

STARTING
1. HORSFALL UNIT
2 REMOVABLE FRONT
3 FEED DOOR
4 INCOMING AIR FILTER
5 OUTGOING AIR FILTER & STACK

RAISING
1 DISEASE-FREE PIGS
2.CLEAN QUARTERS
3.ISOLATION FROM
  DISEASED ANIMALS

FIG 53 1 — Diagrammatic outline of method used to obtain

tor, by reaching into the cavity, lifts the horns of the gravid uterus up to and out of the incision The uterus is cut free by excising through the cervix and is passed quickly through an antiseptic lock into a hooded work table where the pigs are liberated

The pigs are torn from the uterus as soon as it enters the hood This is done by placing both hands over the pig grasping the uterus firmly and tearing it by rotating the wrists and forearms outward The operator checks the nostrils and mouth of each pig wiping off mucus and membranes with a dry towel whenever necessary Their navels which were clamped temporarily with a crocodile clamp are ligated near the body with light cotton cord

The covered work table or hood, within which the pigs are removed from the uterus is illustrated schematically in Figure 53 1 The table is kept under slight positive air pressure by the constant introduction of warm filtered air The working temperature of the hood 100–110° F, is maintained by passing the incoming air over a kitchen stove heating element The liquid lock serves to introduce the gravid uterus into the hood without creating air currents which might introduce infectious agents An improved model of this kind of hood has been described by Underdahl and Young (1957)

The filter medium consists of a rectangular 6 × 12 inch No 50-FG spun glass wool filterdown pad (American Air Filter Company, Louisville, Ky) It removes possible infectious materials from the air introduced into the hood The interior is sterilized before each day of use with formaldehyde gas produced by mixing potassium permanganate and formalin

The pigs are transported in sterile carrying boxes to the isolation units housed in clean, previously gassed rooms (formaldehyde gas from ¼ oz potassium permanganate and one pint of formalin for each 1,000–1,500 cu ft) Attendants wear sterile coveralls and face masks The transfer is made quickly to minimize respiratory exposure Attendants routinely dampen their hands with a mild antiseptic solution when handling the pigs The rooms and isolation units are kept at 95–100° F for the first few days and then may be lowered to 85° F

Even casual diseases of young pigs which have been nursed by their dam may prove serious or fatal to colostrum deprived new born pigs Since pigs which are not nursed by their dams are devoid of antibody, ingestion of colostrum and absorption of antibodies from it is ordinarily essential to the survival of the newborn pig (Nelson 1932 1934 Young and Underdahl, 1950) Survival of newborn pigs which have been deprived of colostrum, regardless of how obtained is based upon successful isolation of the pigs from time of release from their sterile uterine environment until several weeks old Disease control depends upon continued isolation from so called normal swine

The first diet for successful rearing of colostrum deprived pigs was described by Young and Underdahl (1951) It consisted simply of pasteurized homogenized vitamin D milk, egg yolk, vitamin A, and a mixture of inorganic salts Satisfactory results have also been reported by Haelterman (1956) using a similar diet, and by Bauriedel *et al* (1954), and Whitehair and Thompson (1956) using semi synthetic diets

Excellent livability and performance equal to that manifested by pigs suckling their dam can be obtained with colostrum deprived pigs A simple milk egg mineral diet is used as the starting diet A whole egg is mixed into one quart of homogenized pasteurized vitamin D fortified (400 units per qt) milk in an electric blender Five ml of a mineral mixture (19 8 gm $FeSO_4$ $7H_2O$, 3 9 gm $CuSO_4$ $5H_2O$, 3 6 gm $MnCl_2$ $4H_2O$, and 0 26 gm KI in 1 liter of water) are added per quart of milk Vitamin K is supplied by addition of 1 ml of an aqueous solution of Klotogen F[1] (1 gm in 210 ml distilled water, this gives a final concentration of 1 mg of vitamin K per qt) This mixture is brought

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[1] Abbott Laboratories

to boiling, then cooled to approximately 100° F for feeding the first day Thereafter the modified milk is fed without bringing it to a boil Vitamin K is no longer added Pigs are fed morning, noon, and night from shallow flat bottomed pans

Choice of the type of isolation in which colostrum deprived pigs will be reared depends upon several factors Most important is the chance of exposure to other pigs Bauriedel et al (1954) and Whitehair and Thompson (1956) used relatively crude isolation procedures to raise successfully a relatively small number of pigs Similar early isolation was used by Young and Underdahl (1951) for raising their pigs They found, however as the numbers of pigs increased, that more elaborate isolation was essential to success They have developed isolation units satisfactory for exclusion of disease (Young and Underdahl, 1953) Satisfactory early isolation is obtained by less elaborate isolation units described by Haelterman (1956)

When first considered to obtain pigs by hysterectomy, hysterotomy, or cesarotomy and rear them for their first weeks in isolation as a disease control measure seems impractical The number of pigs which can be produced in this manner is small Special equipment and skilled personnel are required In spite of what might be considered discouraging aspects of these techniques, the potential for disease control is enthusiastically presented by several workers who have had experience in this area (Young et al, 1955b, Done, 1955 Hoerlein et al, 1956 Young and Underdahl 1956, Whitehair and Thompson, 1956) Swine populations may be increased quite rapidly so that a few pigs obtained clean can provide, through subsequent normal farrowing, the basis for a sizeable population of clean pigs in a relatively short time

A practical approach to elimination of chronic diseases from our national swine population is presented in the following paragraphs

1 Obtain aseptic pigs These are obtained from good breeding stocks as described above Care should be taken to obtain pigs from dams which were in unquestionably good health the first trimester of gestation Attention to this detail should reduce the chance that some mild agent might infect the pigs in utero and be carried through to birth to infect other pigs (Young et al 1955a Young 1955)

2 Rear pigs in isolation Pigs are housed in individual isolation units from birth by one of the above means until 1 week old During this period they are fed a modified cows milk From 1 week until 4 weeks of age they are preferably housed in groups of 8 to 12 in isolation brooders Less definitive isolation may be used provided it is adequate to prevent exposure of the pigs to infectious agents Fumigation of quarters with formaldehyde gas 24 hours before entry of new groups of pigs is useful During the period from 1 to 4 weeks pre creep feed is made available to the pigs as food in addition to the modified milk Milk is withdrawn at 4 weeks

3 Mature on farms Pigs previously adapted to eating solid feed are placed in groups of 10 to 20 on farms from which all other swine have been removed Ordinary rearing methods are employed except that no new so-called 'normal' stock is introduced, and contact with other swine must be avoided by the farmer Stock is raised to maturity In general control of diseases will be accomplished by isolation There is some risk, however, of introduction of hog cholera virus from unknown reservoirs Since this disease may cause severe mortality among swine at a considerable loss to the producer immunization of swine against hog cholera is advised The recommended method is the simultaneous use of rabbit attenuated hog cholera virus and small doses (10–20 cc) of anti hog cholera serum The attenuated vaccine virus should be of rabbit origin to minimize the possibility of passenger viruses being introduced accidentally from the vaccination Use of attenuated vaccine virus of swine origin presents a continuous hazard as a source of swine virus diseases other than hog cholera This would be

most undesirable and perhaps disastrous in a herd which has been freed of disease by much effort.

4. *Resume normal birth.* The stock which is reared to maturity on farms is kept there and used as brood stock. Normal farrowing is resumed, with precautions to avoid introduction of disease. When additional blood lines need to be added, boars from other farms on the same disease control program are introduced.

5 *Restock other farms.* The clean stock obtained on primary farms by steps 1 to 4 is used to repopulate other farms. These considerations for disease control are observed: (*a*) complete depopulation of swine from the premises *before* introduction of the "clean" stock; (*b*) mechanical cleansing and disinfection of premises following depopulation; (*c*) introduction of stocks only from sources or by means which assure continuation of "clean" stock; (*d*) avoidance of direct contact with other swine by the farmer, and of indirect contact as far as possible. Reuse of feed sacks through small local dealers is undesirable.

At the beginning of 1957, the methods described above were only in the exploratory stages at a few of our experiment stations. Progress has been so rapid and the seemingly insurmountable obstacles have been passed so easily that there undoubtedly will be considerable field application of the methods in the near future. Effective control of virus pneumonia of pigs and atrophic rhinitis appears certain. The application of these methods to swine disease problems, therefore, should prove an exciting step forward.

REFERENCES

BAURIEDEL, W. R., HOERLEIN, A B., PICKEN, J C., JR., AND UNDERKOFLER, L. A. 1954. Selection of diet for studies of vitamin B₁₂ depletion using unsuckled baby pigs. Agr. Food Chem. 2:468.

BARBER, R. S., BRAUDE, R., MITCHELL, K. G., AND BETTS, A. O.: 1955 The eradication of virus pneumonia from a herd of large white pigs at a research station. Vet. Rec. 67 40

BETTS, A. O., AND BEVERIDGE, W. I B: 1953 Virus pneumonia of pigs The effect of the disease upon growth and efficiency of food utilization Vet. Rec 65 515.

———, WHITTLESTONE, P., AND BEVERIDGE, W. I B 1955a. Investigations on the control of virus pneumonia of pigs (V P P) in the field. Vet Rec. 67 685.

———, TAYLOR, J H., AND CAMPBELL, R. C.: 1955b Virus pneumonia of pigs Further investigations on the effect of the disease upon growth rate and efficiency of food utilization Vet Rec 67 661.

DONE, J T 1955 Aseptic delivery and artificial rearing: A technique for disease control in pigs. Vet Rec 67 623.

HAELTERMAN, E O 1956 Practical isolation equipment for baby pigs. Amer. Jour. Vet. Res. 17 129.

HOERLEIN, A B, ADAMS, C H., AND MEADE, R J: 1956. Hysterotomy to obtain "disease free" baby pigs. Jour. Amer. Vet. Med. Assn. 128.127.

———, HUBBARD, E D, LEITH, T. S, AND BIESTER, H E: 1954 Swine brucellosis. Bull Vet. Med. Res Inst., Iowa State College, Ames, Iowa.

MOHLER, J R 1938. Foot and mouth disease. U S D A. Farmer's Bull No. 666

MULHERN, F J.: 1953. Progress report on the eradication of vesicular exanthema. Proc 57th Ann Meet U. S S Livestock San Assn., p 326

NELSON, J. B. 1932. The maternal transmission of vaccinal immunity in swine Jour Exper. Med. 56 835

———. 1934. The maternal transmission of vaccinal immunity in swine. II. The duration of active immunity in the sow and of passive immunity in the young. Jour Exper. Med 60 287.

SHUMAN, R D, AND EARL, F. L.: 1956 Atrophic rhinitis. II. A study of the economic effect in a swine herd. Jour Amer. Vet. Med. Assn 129 229.

———, AND STEVENSON, J. W. 1956 Atrophic rhinitis VI. The establishment of an atrophic rhinitis free herd of hogs Jour Amer Vet Med Assn 128 189

THOMPSON, C M, WHITEHAIR, C R, MacVICAR, R. W, AND HILLIER, J. C 1952. Observations on artifically raising healthy swine Okla. Agr. Exp Sta. MP—27 74.

UNDERDAHL, N. R, AND YOUNG, G A · 1957. An improved hood for swine hysterectomies. Jour. Amer. Vet. Med. Assn. 131 222.

WHITEHAIR, C. K, AND THOMPSON, C M · 1956. Observations on raising "Disease free" swine Jour. Amer. Vet. Med Assn. 128 94.

YOUNG, G. A. 1955 Influence of virus infection vaccination or both on embryonic and fetal development Proc. Book Amer Vet Med Assn 92nd Ann Meet p 577

————, KITCHELL, R. L., LUEDKE, A J, AND SAUTTER J H 1955a The effect of viral and other infections of the dam on fetal development in swine I Modified live hog cholera viruses — immunological, virological, and gross pathological studies Jour Amer Vet Med Assn 126 165

———— AND UNDERDAHL, N R 1950 Neutralization and hemagglutination inhibition of swine influenza virus by serum from suckling swine and by milk from their dams Jour Immun 65 369

————, AND ———— 1951 A diet and technic for starting pigs without colostrum Arch Biochem and Biophysics 32 449

———— AND ———— 1953 Isolation units for growing baby pigs without colostrum Amer Jour Vet Res 14 571

———— AND ———— 1956 Measures to obtain and to maintain a healthy herd of livestock Jour Amer Soc. Farm Managers and Rural Appraisers 20 63

————, ————, AND HINZ, R W 1955b Procurement of baby pigs by hysterectomy Amer Jour Vet Res 16 123

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